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PNEUMOCONIOSIS

THE STORY OF DUSTY LUNGS*

By LEWIS GREGORY COLE, M.D.
Director of Silicotic Research, John B. Pierce Foundation
NEW YORK, NEW YORK

A MORE scientific term for the title of this paper would have been "pneumoultramicroscopicsilicovolcanokoniosis," which is said by the *Reader's Digest* to be the longest word in the English language. Yet, even this term is not thoroughly comprehensive.

Morbid changes in the lung caused by the inhalation of dust have become more of a social, economic, legislative and medicolegal problem than strictly a medical one.

The literature indicates that dust hazards were recognized before Christ but it has become a serious industrial problem only since the advent of technocracy or modern tools, particularly the pneumatic drill and jack hammer.

It was not until about the turn of the century that the dust hazard centered around silica, silicates and silicotuberculosis, and it was not until the second or third decade of this century that silica was considered the main offender and that most other dusts were considered innocuous. Now many observers are directing their attention mainly to the lesions that they consider are due to the inhalation of silica (SiO_2).

The more comprehensive problem of morbid changes in the lung due to the inhalation of various kinds of dust seems to have been overshadowed by the controversy concerning whether only such lesions as are caused by silica should be considered social and economic problems, or whether the morbid changes following the inhalation of other dust should be included in the legislative acts designed for the protection of persons exposed to various dust hazards.

There is even a serious controversy as to whether the silicates are capable of causing morbid changes in the lungs that are of clinical, economic and social significance, or whether the dust must be silica.

Perhaps it would be well to clarify this point by a definite statement; namely, that observers are divided into two distinct groups—one group maintains that silica is the only dust capable of causing morbid changes in the lungs of sufficient significance to become a social, economic and legislative problem; the other group maintains that dust other than silica may predominate in the lungs and that these dust particles and their accompanying morbid changes in the lungs constitute social, economic and legislative problems.

There are two investigating approaches

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that are as diametrically opposed as the above mentioned groups. The first or more conventional approach is to learn all that can be learned from the literature concerning what has been done on the subject and then to correlate those data if possible, and later attempt to prove or disprove such findings as may interest one. The other approach is to assemble all the facts that can be observed without being guided or misguided by observations and conclusions recorded by others.

The first is the conventional approach and I know of no article, certainly no recent one, that so adequately deals with the subject as the one by Pendergrass.* This essay is a masterpiece of medical literature and was presented before the Wayne County Medical Society as a memorial lecture to Dr. Preston M. Hickey. It traces the history of dusty lungs from a period just before Christ down to the present time and in proof of this he submits photographic evidence of titles or excerpts from the text of these historical documents. He quotes extensively from thirty-six different articles and emphasizes his conclusions in italics which render them readily accessible.

Much of the early literature and some of the current literature deal with morbid changes in the lungs due to dust that is not silica; one then might ask, "Why then, have some of the recent investigators picked on silica as the main or only offender?" I certainly urge all who are interested in this subject to read the article by Pendergrass, and if they feel so inclined, to compare it with the data herein assembled.

The second and less conventional approach to the subject is that of observing and recording facts regardless of whether they have been previously observed by other workers, and regardless of their apparent insignificance. Findings so observed, be they clinical, roentgenologic, or pathologic, are like sections of a jig-saw puzzle. They should be true, clear cut, well defined, so that as one begins to assemble them, they will readily dovetail with each other, without having to be forced.

Far be it from me to criticize the conventional approach which is more acceptable to most of those who are studying the subject, particularly those who have written on it. Authors take great satisfaction in having their work quoted and requoted. Intentionally we did not use the conventional method in assembling the material for the story of dusty lungs as recorded in the little blue book published by the John B. Pierce Foundation in the preliminary report on pneumoconiosis (silicosis), nor did we use it in assembling the data for the second edition of this book, which is much more comprehensive than the first and is to be profusely illustrated.

It is only since these data were assembled and described that I have gratified my curiosity as to what other observers have recorded. Again I would urge those of you who are interested in this subject to read articles by recognized authorities such as Lanza, Pancoast, Winslow, Greenburg, Gardner, Pendergrass, Drinker, Sayers, Hatch, Cummings, and many others, particularly the investigators in South Africa, who really started the serious investigation of this subject. After reading these articles you should make an attempt to correlate the observations and conclusions of these men and then set that material up and compare it with that which has been assembled for the second edition of our "Story of Dusty Lungs."

A good example of the second approach is Pancoast's investigation of the roentgenologic and clinical findings in the hard coal miners of the Pennsylvania field. His broad, comprehensive approach is indicated by the title of his first article, namely, "Effects of dust inhalation on the lungs." Indeed, it was his monumental work in this field that started the roentgenologic and medical investigation of pneumoconiosis (silicosis) in this country. About this time (1915) Lanza investigated the subject of

* Pendergrass, E. P. Some considerations concerning roentgen diagnosis of pneumoconiosis and silicosis. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *48*, 571–594.

silicotuberculosis from a statistical standpoint for the U. S. Bureau of Mines, but it was Pancoast who started the clinical and roentgenological investigations, and it was he who established roentgenology as a method of investigation in the problem of dusty lungs throughout the world.

In the late teens or early 1920's Pancoast became the leader of a group of men who were interested in this subject, but in the late 1920's and early 1930's a line of cleavage became apparent between those who followed the comprehensive approach of Pancoast, on the one hand, and those who believe that silica is the only offending dust, on the other.

At a meeting at Lake Placid in 1934 the line of cleavage split, and a definition of silicosis was adopted that rejected the "peribronchial perivascular lymph node manifestation" of Pancoast. From hearsay I understand that there was considerable controversy concerning this definition. It has been said that there were nearly as many definitions as there were men present, and that in reality the definition was a compromise. Some of those who participated in formulating the definition, or at least who sanctioned it, were not physicians, and it is evident that the majority of those present were not in sympathy with the qualified roentgenologist, as is evident by the declaration of their object, which is as follows: "A diagnosis may be required of a physician in a plant, in a public health office, in a sanitarium, or in a physician's private office. Sometimes he may have had little experience in the interpretation of roentgenograms of the chest and will be forced to lean heavily on the interpretation of the roentgenologist. The legal aspect of the problem has placed great responsibilities on the medical profession and has been a source of embarrassment. Only a physician who has examined the subject, has obtained occupational history of adequate exposure to silica dust [what is adequate?], and has before him a suitable roentgenogram of the chest [which it is admitted he is not qualified to interpret], should make a diagnosis of silicosis." The roentgenologist not in possession of these facts can merely state whether the shadows which he sees in the roentgenograms are consistent with this diagnosis. The vast majority of these cases, particularly in the early stages, have absolutely no symptoms, no physical signs and nothing on which the clinician is justified in making the diagnosis of silicosis except possibly the history of exposure to dust. Therefore the diagnosis is based fundamentally on the roentgenologic findings, and why should it not be a roentgenologist who is to interpret these findings?

They further state that, "If objective terms descriptive of the type of pathological changes could be generally adopted material progress would result. The clinician would not have to accept a diagnosis from the roentgenologist, general students of the disease would be able to correlate the findings of the various observers and more accurate definition of roentgenograms would be available for medicolegal purposes. Roentgenograms of the chest, which are notoriously difficult to reproduce for illustrations, should be described in word pictures capable of interpretation in the light of the personal knowledge of the observers." In other words, the clinician not experienced in the interpretation of roentgenograms could put his own personal opinion into the record and not subsequently be embarrassed by having to face up to prima facie evidence as shown in the roentgenogram and interpreted by one capable of such interpretation

It is generally accepted that roentgenograms form the fundamental basis for the diagnosis of morbid changes in the lungs resulting from the inhalation of dust, regardless of what terms are employed to designate the lesion. Interpretation of roentgenograms has developed into one of the highest specialized fields of medicine, and yet it was the object of this clinical and lay groups to limit the disease by a specific definition so that the industrial physician, public health officer and the physician in his own private office, who may have had

little experience in the interpretation of roentgenograms of the chest, will not be forced to lean heavily on the experienced roentgenologist for a diagnosis of this condition, and for aiding in the solution of the social, economic and medicolegal problems, which arise therefrom.

Instead of simplifying these problems this definition has tended to make them more complicated. According to this definition, it takes ten or fifteen years of intensive exposure for one to develop silicosis. What of that group of Gauley Bridge workers, many of whom were dead in two or three years from the time of their first exposure? If it requires a history of adequate exposure to dust, what constitutes an adequate exposure both in quality and in quantity? What of the men who died without having developed typical nodulation, as defined by this definition? What of the men who had large multiple masses in the lung with very few nodules? Are all of these persons to be deprived of compensation because they were unfortunate enough to develop a manifestation of pneumoconiosis (silicosis) that failed to conform to so simplified a definition that the inexperienced industrial physicians, health officer and private physician could make the diagnosis without the aid of an experienced roentgenologist?

Since this definition was written much of the literature by those who participated in making it has been devoted to the differentiation of the unusual cases from the classical ones, as described in this definition, or they have vied with each other in coining such terms as "so-called silicosis," "pseudosilicosis" "modified silicosis," and the like. The definition previously given, and a later statement by Gardner in an elaborate article, specifically eliminates Pancoast's peribronchial perivascular lymph node manifestation of pneumoconiosis: "Insufficient exposure to silica may cause a certain amount of exaggeration of the linear shadows cast by the vascular tree and enlargements of the tracheobronchial lymph nodes. These changes are not specific of silica; they may be produced by inhaling other dusts, by infection, by heart disease. Since all these factors may be operative in some one person who worked in a silica industry, one is not justified in attributing the roentgenological appearance to silica." Let it be emphasized that without nodulation a diagnosis of silicosis is unwarranted at the present time.

After my early observations had convinced me of the value of Pancoast's work, I met him while I was demonstrating an exhibit at Atlantic City. He visited it and when I demonstrated to him our pathological findings which substantiated his roentgenological findings, he was pleased. I said to him, "What happened in 1934? Did your crew mutiny and throw you overboard?" He smiled as best he could, grasped my hand, and said, "Cole, you always could read between the lines. I'm tired. Goodbye Cole." That was the last time I ever saw him. The wonderful work, particularly on the peribonchial perivascular lymph node manifestation is still being repudiated, even by those who were closest to him.

Now as we approach the broad problem of the morbid changes in the lungs resulting from the inha'ation of various types of dust and their clinical and roentgenologic manifestations by the unorthodox approach, we will do well to follow Pancoast's example, and make an intensive study of roentgenograms which are accompanied by as adequate a clinical history and physical examination as it is possible for the attending physician to give. Hundreds of such roentgenograms from many communities in this country and Canada of subjects who have been exposed to dust in many different industries are on file at the John B. Pierce Foundation, and any physician is not only permitted but is invited to study them. At the beginning of this unconventional approach, may I ask you to wipe the slate clean, if perchance you can, forget the definition of silicosis, and the controversy as to whether silica (SiO_2) is the only dust that causes morbid changes in the lungs worthy of considera-

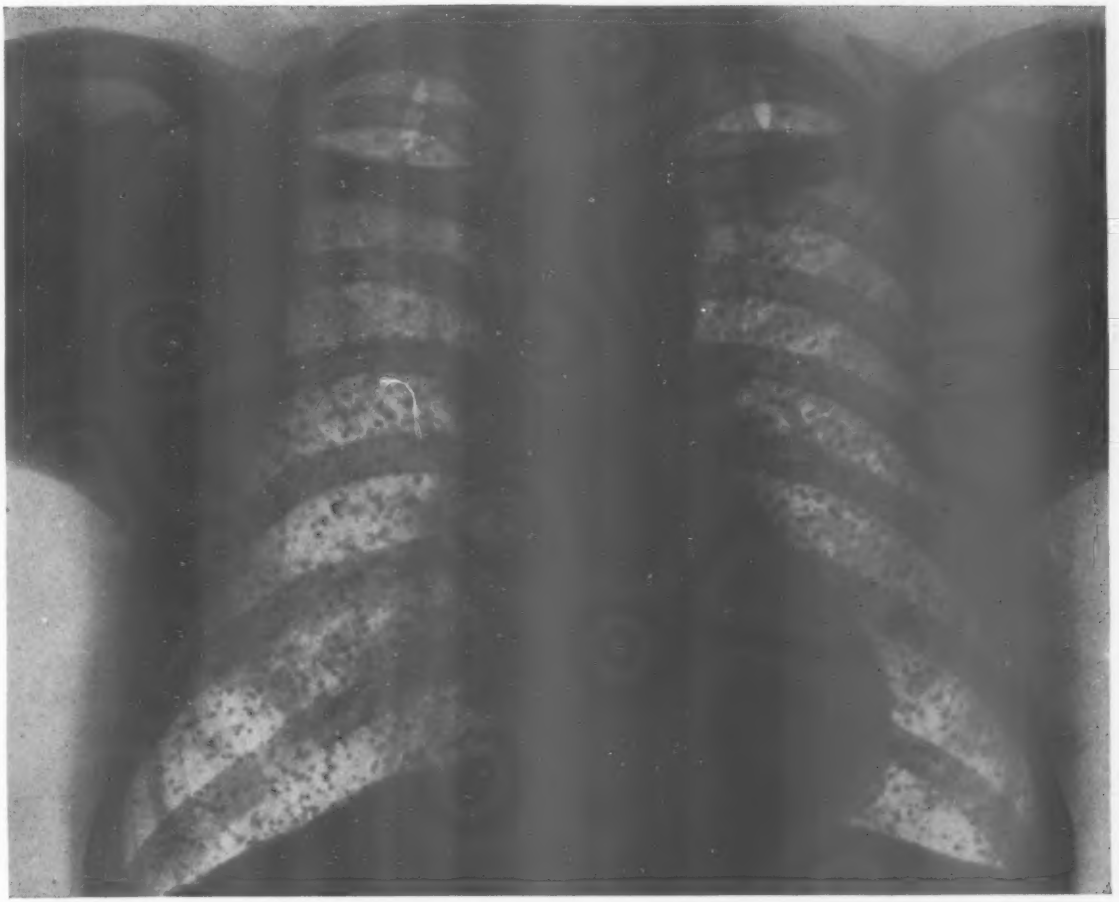

Fig. 1. (Lent by Dr. Hill, New York.) Bilateral symmetrical mottling composed of small round spots with clear cut, well defined edges. These spots are separated from one another by well ventilated lung, and bear no constant relationship to the blood vessels and bronchi as they radiate from the hilum.

Occupational and Clinical History. Patient, aged forty-eight, weight 125 lb., height 5′4″; worked since nine years of age. Until 1924 he did odd jobs and for a short period was a moulder's helper; 1924 to 1928 worked in a mixing room of an asbestos department; 1928 to 1936, as a WPA or PWA employe; 1936 to 1942 worked as a clay mixer in both wet and dry mix.

He has had coughing spells for many years which he says is better when he has good food and sleep; has no dyspnea or any other symptoms.

Social and Economic Status. He can work and wants to work.

Since the patient has inhaled a variety of dusts in different industries, who can say which dust caused these characteristic findings?

tion, or whether silicates may also cause these morbid changes in the lungs. Indeed, the problem of presenting this material would be greatly simplified if you would start at the beginning, check off paragraph by paragraph, illustration by illustration, the data herein submitted, and mark on the border of the page either approved or disapproved, or questioned, and then analyze the conclusions, and mark down whether they are rational or irrational. Do this without attempting to correlate these observations or conclusions with the controversial problems so adequately presented by Pendergrass in his conventional approach, or with the observations and conclusions of others. However, you are asked to refrain from doing this until you have followed me through to the bitter end; then you are urged to check these data and

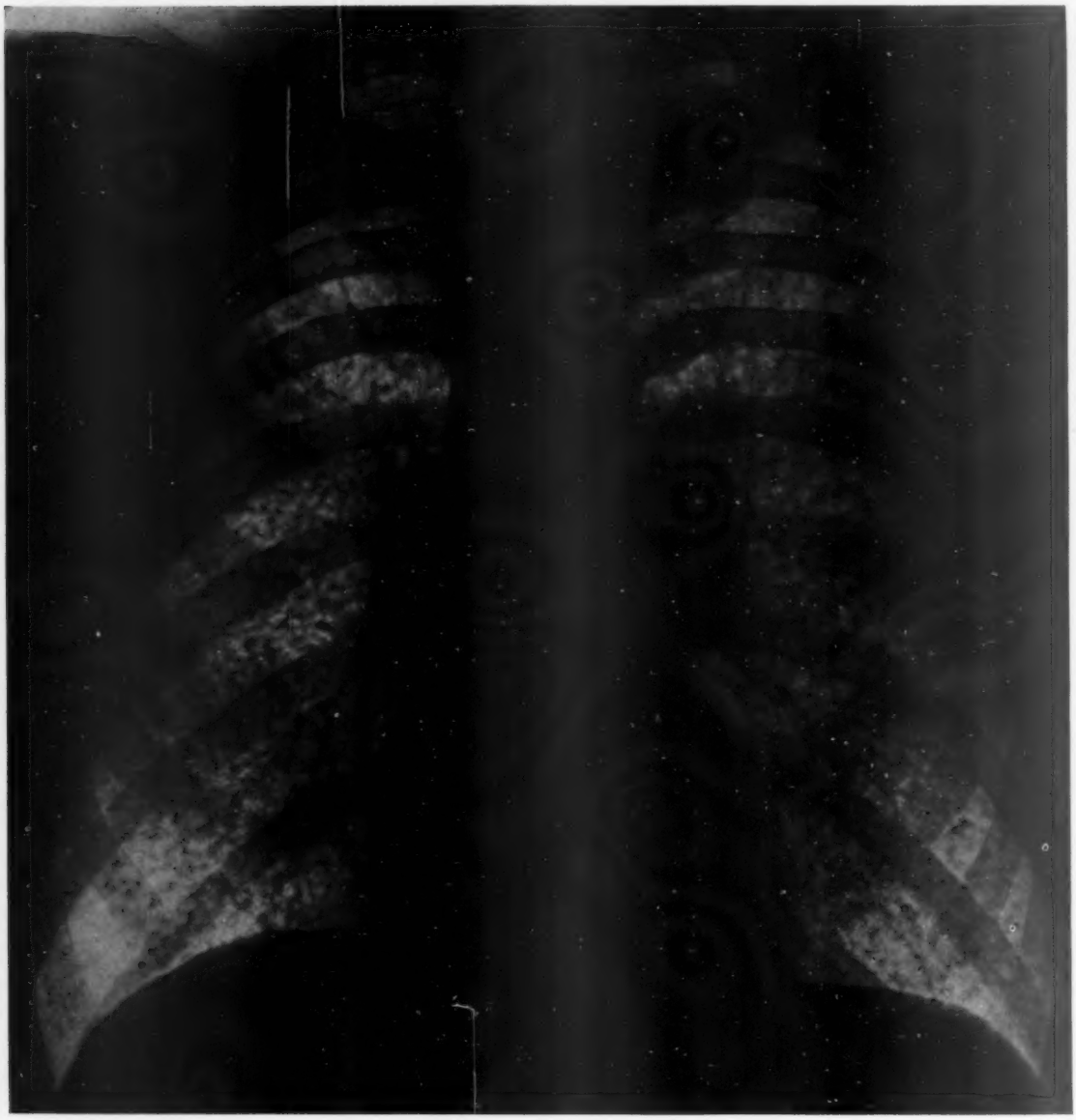

Fig. 2. (Referred by Dr. Roberto, Yonkers, N. Y.) *Roentgen Findings.* Bilateral symmetrical mottling with small irregular shaped spots that tend to join each other. The shadows are not so clear cut as those shown in Figure 1, and they bear a definite relationship to the smaller blood vessels and bronchi as they radiate toward the periphery; the shadows at the hila are moderately accentuated. There is diminished ventilation of the mid lung fields compared with the bases and apices. When these findings are compared with those of Figure 1 their differences are distinctly discernible.

Occupational and Clinical History. Male, aged thirty-five. First exposure to dust at age of sixteen; he then worked without a mask for seven years as a dry and wet driller in mines with apparently high silica content. For the last twelve years he has had very little exposure to silica dust. He has gained weight from 130 to 153 pounds since quitting underground work. He has had some dyspnea during the past seven or eight years which did not develop until four or five years after he ceased his underground exposure to silica. It does not interfere with his work, which is now not manual labor. He plays golf on flat courses but hilly courses bother him, as does swimming.

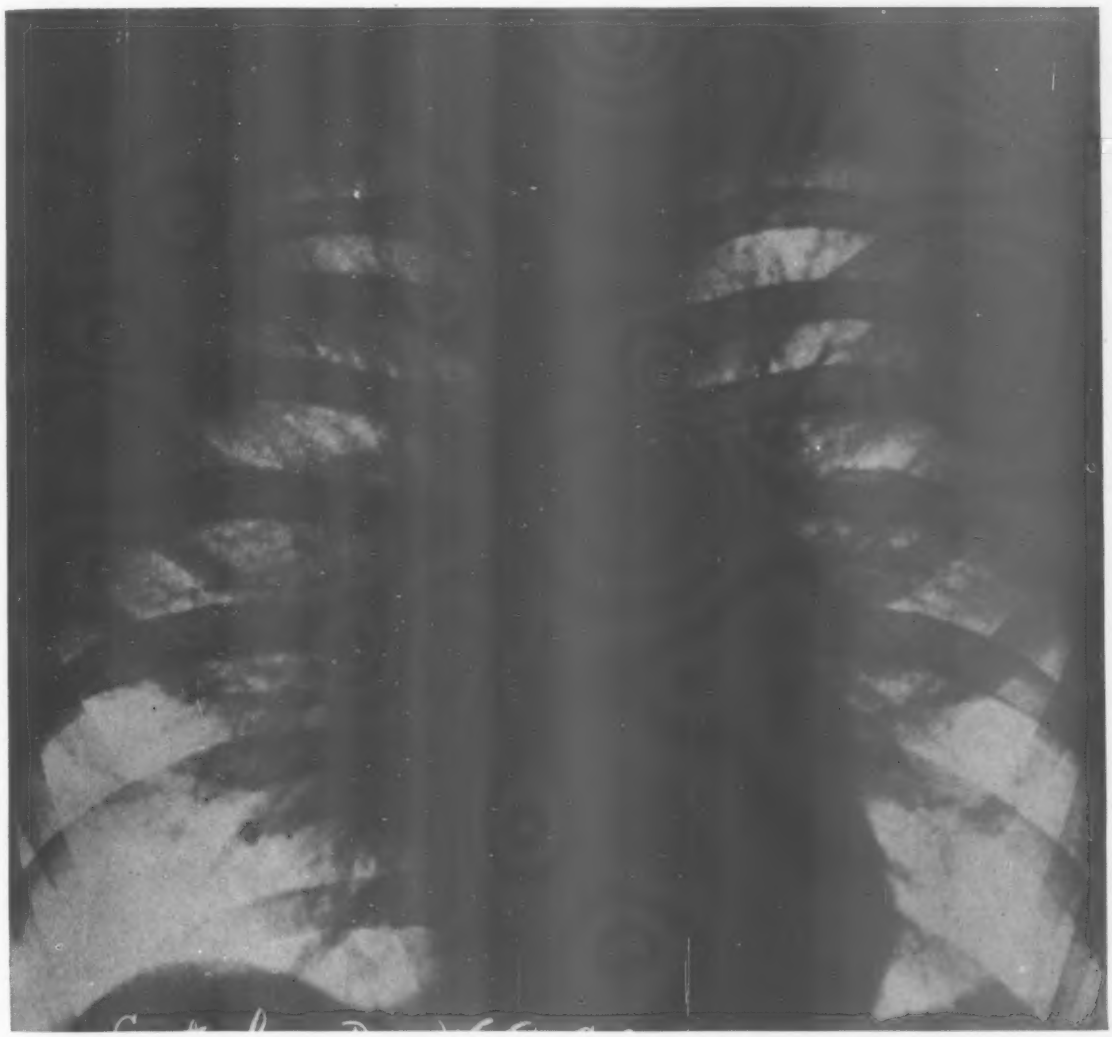

Fig. 3. *Roentgen Findings.* Very extensive accentuation of the hilar and linear markings, which is bilateral and relatively symmetrical, radiating out about as far as the middle thirds; diminished density in the mid lung fields with marked emphysema at both bases. There is little, if any, nodulation.

Occupational and Clinical History. Buffer and grinder for twenty years, using carborundum wheel. No dyspnea, cough or other symptoms.

Social and Economic Status. He could work, wanted to work, and resented being examined at routine examination lest he should be excluded from work.

compare them with those from any or all other sources.

Unquestionably roentgenograms form the basis for diagnosis of morbid changes of the lungs incident to the inhalation of dust, regardless of the dust inhaled or the term that may be applied to this lesion. If one were to observe hundreds of roentgenograms assembled from various sources, he would be impressed with the wide variety of roentgen findings. The vast majority of these roentgenograms which show morbid changes in the lungs are obtained because of routine voluntary or compulsory examination of subjects exposed to dust hazards. As indicated by the clinical history and physical examination accompanying these roentgenograms, most of the subjects have few, if any, symptoms and have very few physical signs. In some of the cases, roent-

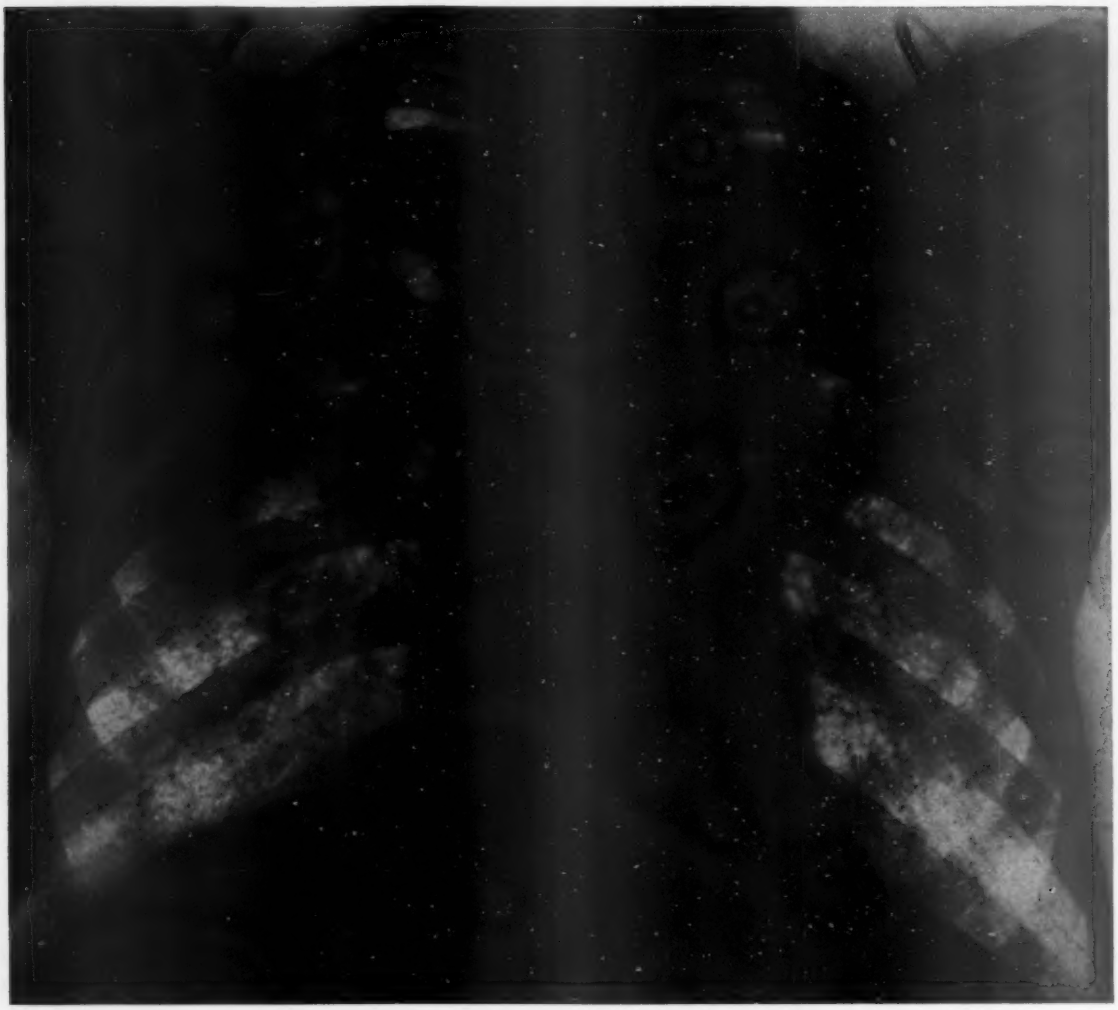

FIG. 4. (Lent by Dr. R. E. Pound, New York.) *Roentgen Findings.* Massive, dense areas of the lungs involving both upper lung fields well out toward the periphery, but the apices above the clavicles are singularly clear. The lower mid lung fields are mottled with a fine irregular pattern that bears a relationship to the smaller blood vessels and bronchi, and is very similar to the findings observed in Figure 2.

Occupational and Clinical History. Male, aged forty-five; had twelve years' exposure to iron ore mines. Light to moderate dyspnea and some cough. The massive areas of density in the upper mid lung fields is out of proportion to the history of exposure, but the fine mottled appearance in the lower lung fields is similar to that observed in iron electric welders.

genograms coincide so accurately with the definition previously quoted that they could be easily interpreted by one who has relatively little experience. But the majority of these cases differ greatly in one respect or another from the accepted definition of silicosis.

From the moment the roentgenogram is made of a subject who has been exposed to a dust hazard, if it shows some variation from the normal findings, that subject becomes a potential social, economic and industrial problem, regardless of whether the findings are slight or extensive. A few roentgenograms with both clinical history and roentgenologic findings incorporated in their legends, are selected from hundreds to reveal the great diversity of roentgen findings and the ramifications of the social and economic problem (Fig. 1–7).

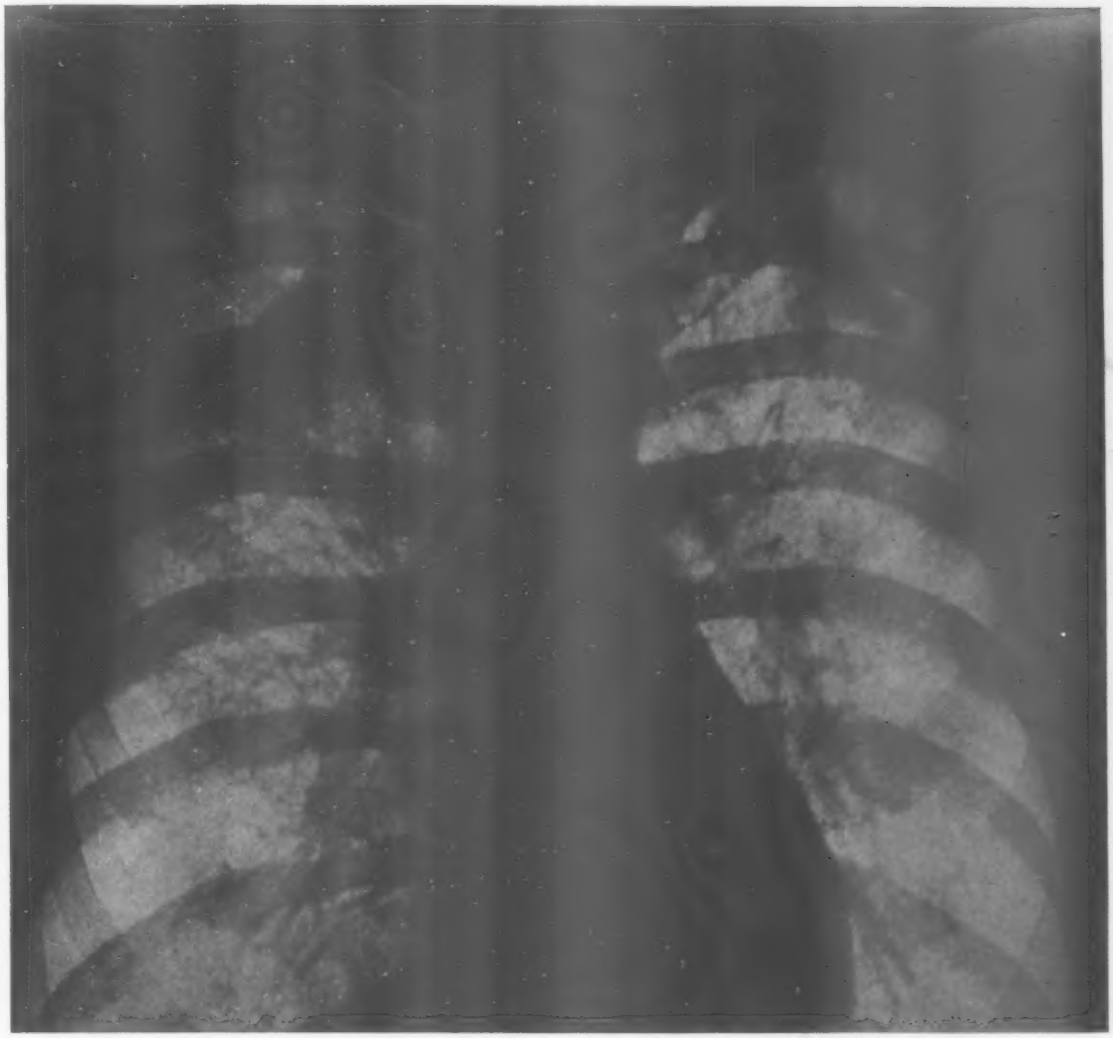

Fig. 5. (Lent by Dr. Eugene P. Pendergrass, Philadelphia.) *Roentgen Findings.* Bilateral, relatively symmetrical, smooth, homogeneous shadows involving both upper mid lung fields. "Angel wings" sign: The dense areas have sharp, clear cut upper borders but they feather out below, grading off into the normal lung density. The lung structures in the apex above this shadow are singularly normal and there is very little mottling in the lower mid lung fields. This finding is very characteristic of a dust lesion, and is due to an accumulation of dust, dust laden phagocytes, and collagen beneath the pleura at the apex of the *lower* lobes, in contradistinction to the apex of the upper lobe. This finding reminds one of rudimentary wings folded up on the back, and we have referred to it as the "angel wings" sign. No clinical history of this case is available.

An intensive study of these seven roentgenograms (Fig. 1–7), or as large a series as the reader has available, should stimulate one's curiosity as to what morbid changes in the lungs cast the great variety of roentgen shadows to which we have just referred.

Pancoast was curious concerning the morbid changes in the lungs which cast the roentgenologic shadows which he so clearly described, but evidently he could not get the cooperation of the pathologists that he desired in his own hospital, that is, the Hospital of the University of Pennsylvania Medical School, because in 1935, I searched the pathology files of this institution and

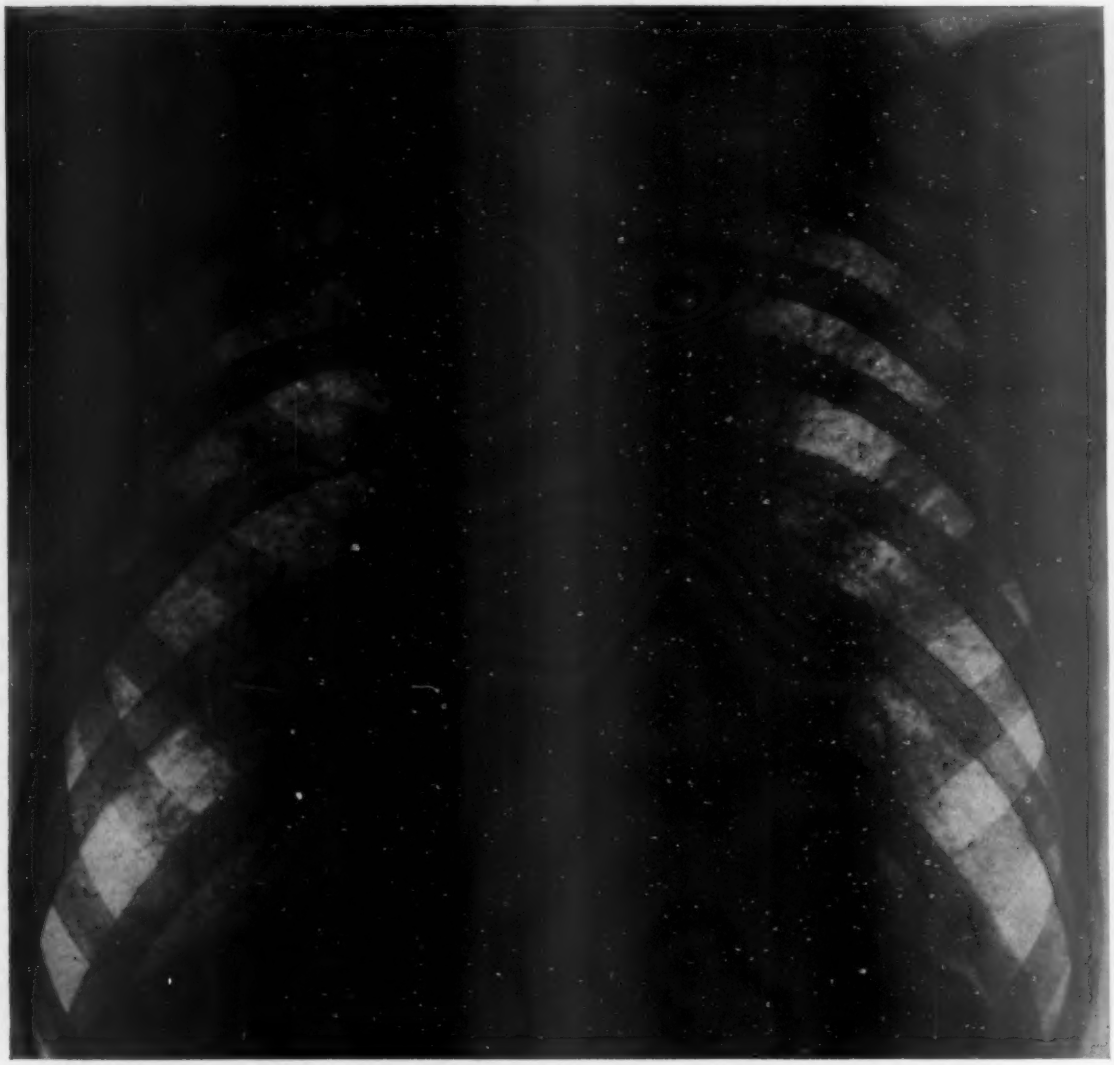

FIG. 6. (Referred by the late Dr. I. Seth Hirsch, New York.) *Roentgen Findings.* Bilateral, relatively symmetrical mottling, with very fine, indefinite spots that are almost identical with the findings observed in the case of seven years' exposure to silica as described and illustrated in Figure 2.

Occupational and Clinical History. Male, aged twenty-five; worked in a factory where his only exposure to silica was an occasional grinding of machine tools and a single exposure to emery dust which was blown into his face by a gust of wind through an open window. He has real, extreme dyspnea, clubbing of the fingers and deformity of the finger nails.

Social and Economic History. The patient sued for $75,000 and settled for $7,500. After the settlement he was intensely studied under my supervision. Slightly enlarged axillary lymph nodes revealed microscopic findings interpreted by several pathologists familiar with sarcoids as being characteristic of this disease. Other pathologists questioned this diagnosis and even doubted the existence of such a disease as a pathological entity.

This is inserted to illustrate those lesions of the lungs which resemble pneumoconiosis (silicosis) but where the clinical and pathological data do not support the roentgen findings.

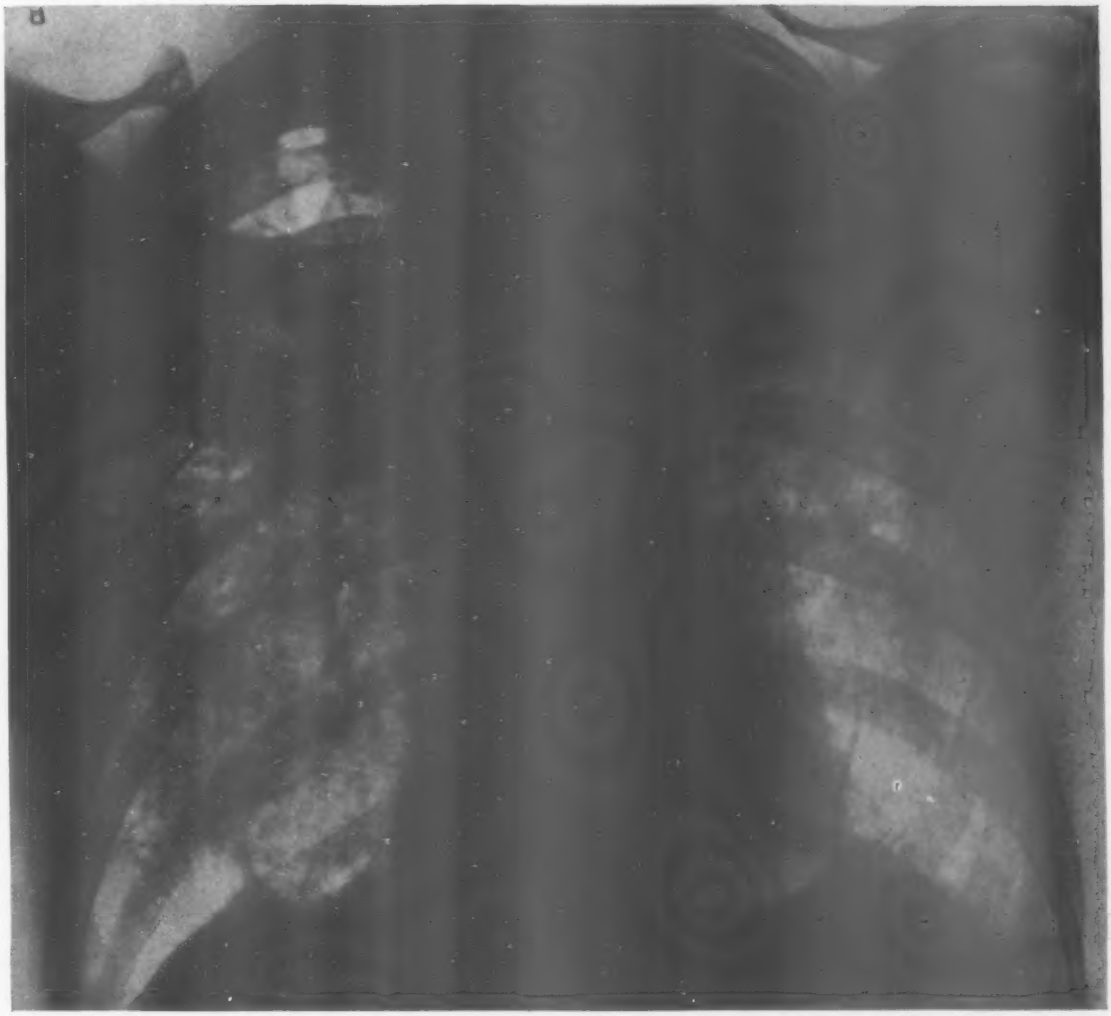

Fig. 7. (Referred by Dr. Francis Ryan, Valhalla, N. Y.) *Roentgen Findings.* Bilateral, symmetrical mottling with spots of irregular shape that are disseminated and separated from each other in the lower mid lung fields, and form relatively large dense masses in the upper mid lung fields, more advanced on the left than on the right. There is a tenting of the diaphragm on both sides, with obliteration of the costophrenic angle on the left. The outline of the heart is markedly obscured even in the region where the lung is not very dense.

Occupational and Clinical History. Male, aged sixty-six. He was literally born and brought up in a dusty atmosphere with high silica content. His father cut granite street paving blocks by hand in his own backyard in England. At the age of sixteen he became a stone cutter's apprentice, and on his first job he helped to cut a stone in a step that was just a mile above sea level. He worked constantly for fifty years as a granite cutter, without a mask and without a ventilating system except for the last four or five years. He definitely maintains that he was not short of breath during this time, and this was confirmed in part by the fact that he was able to work, and at sixty-five he stopped working only because of some Union complications. About three years ago his dyspnea came on suddenly while he was mowing his lawn. The attack was as sudden as a coronary thrombosis. He has been under my personal observation for about three years, his dyspnea gradually progressing. It has periods of exacerbation and is definitely relieved by intravenous injections of 10 cc. aminophylline. He has some cough but very little expectoration; no elevation of temperature; sputum was negative for acid-fast bacilli, and guinea pig inoculation was negative.

FIG. 8. The great variety of roentgen findings in the previous seven illustrations should stimulate one's curiosity concerning the microscopic characteristics of the inhaled dust and the morbid changes in the lung associated with it, as shown in this and the following illustrations. The story of dusty lungs is very briefly and concisely told in the running legends describing the illustrations.

In this illustration, opaque non-refractive airborne flecks that vary in size and shape are seen in the upper part of the field. Similar flecks may be clumped together and form large black flecks, as seen in the lower part of the field. These are not silica because (cont. in Fig. 9)

found only two microscope sections indexed under the heading of silicosis. Both of these were reported as anthracosis, which, as a matter of fact, they were. Therefore Pancoast went elsewhere to find a pathologist who could tell him what morbid changes caused the accentuated linear markings in his group of anthracite coal miners. The one he selected not only failed to identify these morbid changes which cause the peribronchial perivascular lymph node manifestation which he had observed, but he caused Pancoast's crowning achievement of a long and useful career to be repudiated.

Even at the present day Pendergrass recognizes the need for cooperation with various specialists. In his article in the November, 1942, issue of this JOURNAL (page 577) he states, "An ideal program for the diagnosis of pneumoconiosis in the living would include a study of a group, the members of which would be a general physician, a rhinologist, a bronchologist, a clinical pathologist, a specialist in tuberculosis, an engineer interested in industrial hygiene and a general roentgenologist Such a program is too ambitious for practical purposes. . . ."

The roentgenologist is surely in need of a pathologist to aid him in the interpretation of his roentgen findings in terms of pathology, unless he chooses to do this himself.

For the roentgenologist who may be curious to know the morphological characteristics of the inhaled dust and of the morbid tissues in the lungs of those who have inhaled quantities of dust and who are desirous of examining microscope sections of lungs of such cases, or who may be fortunate enough to have the cooperation of a pathologist, an attempt will be made to describe and illustrate gross, macroscopic and microscopic characteristics of these lesions.

A casual macroscopic and microscopic examination of large microscope sections of the lungs of subjects who have been exposed to various dust hazards reveals that the morbid tissue is composed of: (a) deposits of dust, some free in the alveoli, others clumped together in phagocytes either in the alveoli or in the stroma of the lung; (b) masses of collagen laid down in various patterns; (c) cell proliferation; some fixed, some wandering phagocytes; (d) air cysts, caused by the dilatation of the terminal air passages and alveoli; and (e) intravascular deposits of debris and young and aging connective tissue cells.

A more intensive study of these microscope sections leads one to consider these

findings under separate headings: (1) lung dust; (2) phagocytosis; (3) collagen versus connective tissue; (4) six types of pneumoconiosis, and (5) vascular changes.

PNEUMOCONIA (LUNG DUST)

The term "pneumoconia" is not conventional, but there is ample precedence for its use.

Man inhales dust with every breath he takes from the cradle to the grave, sometimes little, sometimes much. Some of this dust is presumably caught on the baffle plates in the upper respiratory tracts; some on the cilia of the trachea and the larger bronchi, and wafted back; whereas other particles of dust are deposited in the terminal air passages and alveoli. Even some of the dust that is deposited in the alveoli is undoubtedly coughed up. The reason for concluding this is the manner in which iodized oil, which has reached the terminal air passages and alveoli, is evacuated from these regions within a very short time.

Such dust particles as may be retained within the lung can be observed microscopically either by the conventional light field method of examination, or by fields rendered dark by cross polarization.* Some

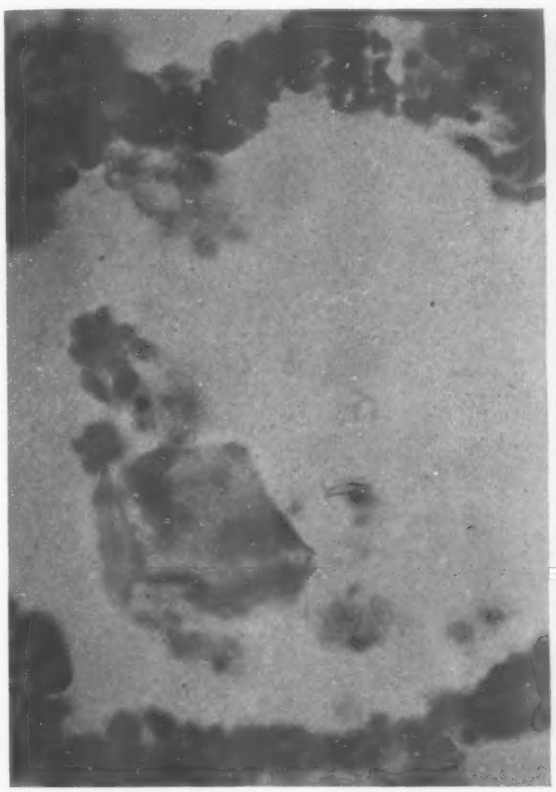

FIG. 9. Silica particles are transparent; they are clear as crystal; only a very large one that looks like an ice cube is visible, in the light field, but when (cont. in Fig. 10)

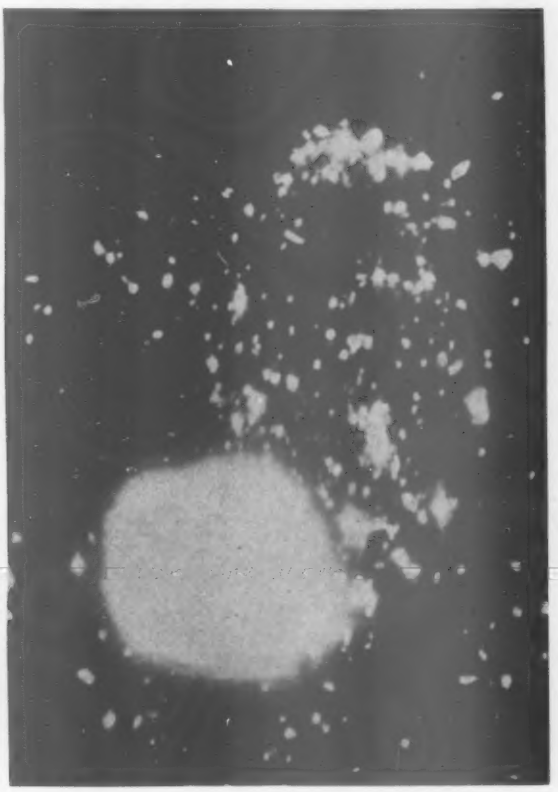

FIG. 10. The same silica particles are viewed in a field rendered dark by cross polarization, they become exceedingly brilliant, reminding one of the moon and stars, but "all is not silica that glitters."

* Cross polarization is a procedure familiar to petrologists but perhaps not to all pathologists, or at least not routinely used by them. The procedure consists of inserting a pair of polaroids or Nikol prisms, one above and the other below the microscopic section. When these polaroids or prisms are parallel, the field appears as the conventional light field, but when they are rotated at right angles to each other, the field is rendered dark and transparent dust particles that have the properties of refraction, refract the light so that each dust particle appears as a brilliant object in a dark field. This procedure is very important in the study of dust particles, and is particularly valuable in the study of such particles lodged in the lungs. It is fully described in our complete text in a chapter devoted to technic.

dust is air borne, some is blood borne, and some may even be of blood origin. Some of the dust particles are opaque, some translucent, and some transparent. Opaque air borne dust particles, as observed in the conventional light field, are often black or reddish brown, or translucent like a shell (Fig. 8). They differ greatly in size and shape, whereas opaque blood borne dust particles are small, uniform in size and relatively round. Other particles of air borne dust are transparent (Fig. 9); they are as clear as glass and are barely seen by the conventional light field method of examination. One would scarcely suspect their presence when viewed with either the low or high power magnification. Only by observing Burle's line around these flecks can one determine their size and shape. But when these transparent particles are viewed in a field rendered dark by cross polarization they appear as brilliant as the stars in the heavens on a clear moonless night (Fig. 10). If one is not familiar with these findings, such a microscopic field is quite startling.

FIG. 11

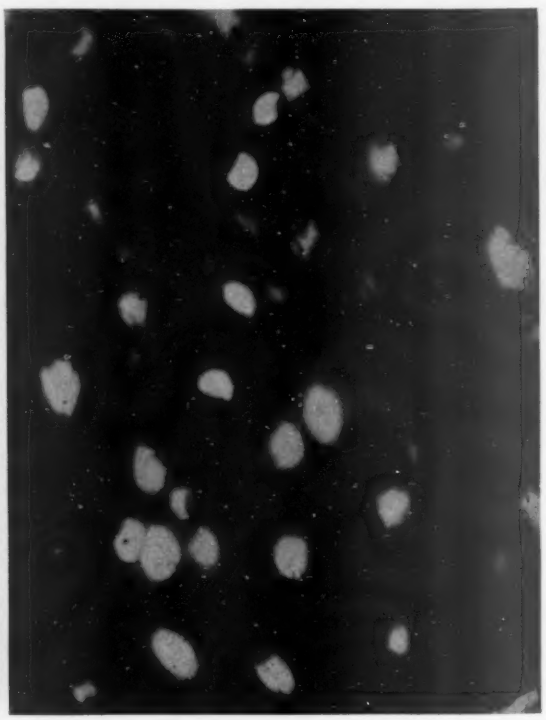

FIG. 12

FIG. 11. Hemoconia in a field rendered dark by cross polarization is as brilliant as silica, but they are small and uniform in size as compared with two or three silica crystals in the upper right hand corner.

FIG. 12. Red blood corpuscles under certain conditions may become birefractive and appear as brilliant spots in a field rendered dark by cross polarization. Such red blood corpuscles can be broken down by heat into four, six or eight fragments, and these are not distinguishable from hemoconia. These correspond in size and shape with the red blood corpuscles except for the fact that some of the fragments have a piece bitten out, so that they resemble a partial eclipse of the moon.

These birefractive particles that are brilliant in a field rendered dark by cross polarization may be silica (SiO_2) or the silicates, but "all is not silica that glitters." Indeed, all that glitters is not even air borne dust. Microscopic fields rendered dark by cross polarization may show quantities of hemokonia that so closely resemble silica that it

may be mistaken for it (Fig. 11).

Hemoconia is defined in Gould's *Medical Dictionary* as follows: "Minute, colorless, highly refractive, spheroidal or dumbbell shaped bodies, constantly present in normal and pathological blood. They are not more than 1μ in diameter, and present active but limited molecular motility, not true ameboid motion." The distinction between small individual particles of silica or silicates and hemoconia is one of the problems of pneumoconiotic pathology, and is discussed in great detail in our complete text.

Sources of Hemoconia. The source of hemoconia is problematic. Red blood corpuscles under certain conditions may be as brilliant as silica in a field rendered dark by cross polarization. These red blood corpuscles, however, are relatively uniform in size, often with a piece bitten out of the side, so that they remind one of a partial eclipse of the moon (Fig. 12), and they are broken down into fragments by heat, whereas silica is not. These broken down fragments are not distinguishable from hemoconia. This subject has been discussed in the text of our book, perhaps to a greater extent than the subject deserves.

In our complete text the characteristics of these transparent, birefractive flecks have also been discussed in detail, but herein they are only mentioned: (*a*) relatively flat crystals have a sudden complete period of extinction as the microscopic field is rotated; (*b*) in multiple granular particles part of the dust particle disappears in one stage of rotation of the field, whereas another part becomes brilliant; (*c*) inclusions, that is black flecks within the transparent crystal; (*d*) lines of cleavage on the surface of the crystal, and (*e*) various shapes of the fragments of the crystal are all microscopic characteristics of transparent birefractive particles that may be either silica or silicates, or hemoconia.

The manner in which these opaque, nonrefractive and transparent birefractive dust particles are deposited in various regions and structures of the lung are illustrated by a score or more of photomicrographs in our complete text, but only four are used to illustrate this text. Hundreds of photomicrographs of the lungs of subjects who have been exposed to intensive dust hazards are on file in the laboratory of the John B. Pierce Foundation. The original microscope sections from which these kodachrome photomicrographs have been made are also on file in the laboratory, and the region in the microscopic field from which these kodachromes are made are recorded on each kodachrome, so that anyone, be he critical or otherwise, is welcome to come to the laboratory, bring his own petrographic microscope and check the observations herein recorded and evaluate my conclusions.

PHAGOCYTOSIS

We have guessed how the air borne dust enters the lung and how some of it has been coughed up; now let us see what happens to that part of the dust that temporarily remains deposited in the terminal air passages and alveoli.

Dust of air borne and blood borne origin and also bacteria deposited within the alveoli seem to stimulate phagocytes to emerge from the blood stream and engulf dust and other organic substances lodged within the alveoli. Many of you have seen microscopically the circulation of blood either in the window of a rabbit's ear or in the omentum of one of the lower animals, as it is spread out over the microscopic field. Those who have not seen the original circulation of the blood may have seen motion pictures revealing the circulation. As will be recalled, the blood passes through the capillaries not as a constant stream, but in fits and starts, in unison with the heart beat. Phagocytes circulating in the blood stream may first cling to the inner wall of the capillaries and then emerge through chinks in the capillary walls. They then wander around in the adjacent structures and later they either wander off into the lymph stream or reenter the blood stream through other chinks in the alveolar wall.

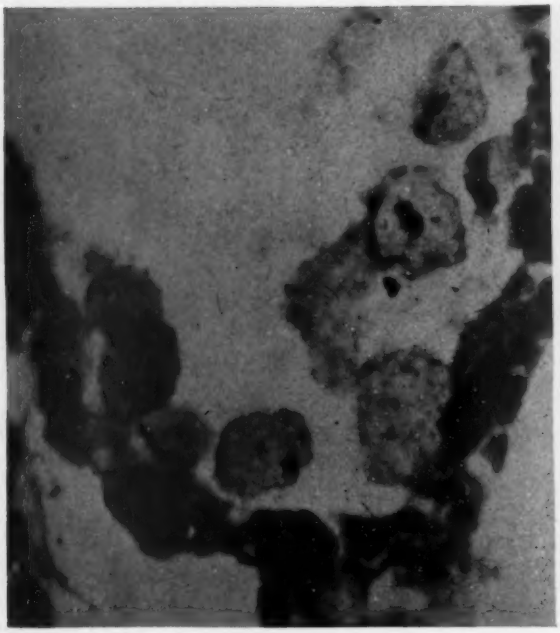

Fig. 13. Phagocytes emerge from the walls of the alveoli and mop up dust, sometimes little, as seen in the upper right field, sometimes much, as in the lower left field. This opaque dust is not silica, but (cont. in Fig. 14)

Fig. 14. Silica and hemoconia are observed when the same field is rendered dark by cross polarization; they appear as white spots on a black background.

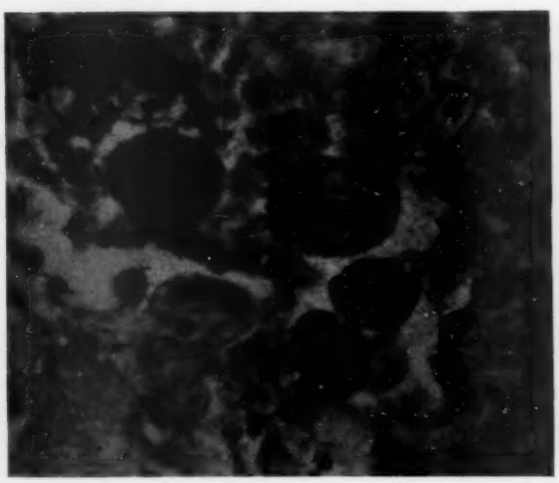

Fig. 15. If a phagocyte mops up only black dust, which is not silica, it becomes a "black phagocyte"; sometimes it takes up so much that its nucleus is obscured, or the nucleus may appear as a "red eye," as shown to the right.

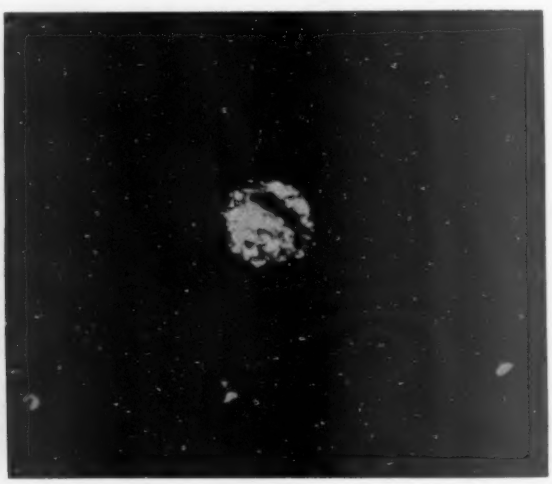

Fig. 16. If a phagocyte mops up a preponderance of birefractive particles, silica, the silicates or hemoconia, it becomes a refractive phagocyte; when viewed in a field rendered dark by cross polarization the nucleus may be obscured and the phagocyte killed by its overload, or (cont. in Fig. 17)

I do not know of this experiment having been done on the lungs of humans or even on the lungs of lower animals, and it is hard for me to conceive how it could be accomplished, but one may reasonably conclude that this physiologic process known to occur in the omentum and in the rabbit's ear also occurs in the lungs of lower animals, or even in man. An intensive study of the phagocytes observed in the microscope sections of these dusty lungs seems to confirm the physiologic phenomenon just recorded. In these large microscope sections some phagocytes, as observed by the conventional light field method, contain opaque, black, brown or translucent particles. It must be borne in mind that these particles are not silica because silica and even the silicates are relatively transparent and are scarcely visible in the conventional light field.

If we now study some of the phagocytes that have recently emerged from an alveolar wall, it will be seen that some are relatively free of these opaque dust particles. Others contain a goodly number, whereas still other phagocytes in the same alveolus may be literally filled with these opaque particles that are not silica (Fig. 13), because silica particles are so transparent that they are not readily visible in the conventional light field. Only by a careful scrutiny can one see the Burle line surrounding the transparent dust particles.

When the same field is rendered dark by cross polarization, many transparent birefractive particles that were not visible in the light field become brilliant in the dark field (Fig. 14). Some of these are silica, some are silicates, and others are hemokonia.

If a phagocyte ingests only black flecks, which are not silica, it becomes a "black phagocyte" (Fig. 15). Its nucleus may be visible and appear as a "red eye," or its nucleus may be obscured by its overdistention with black particles. On the other hand, the phagocyte may take up mostly birefractive particles. It then becomes a birefractive phagocyte (Fig. 16), as observed by cross polarization, or "white phagocyte" in contradistinction to the "black phagocyte." Some of the phagocytes are apparently killed by their overload of transparent, birefractive particles. As the organic material within the dead phagocyte is absorbed, the refractive particles are redistributed on the lining membrane of the alveolus; that is, if the phagocyte is still retained within the alveolus when it dies.

Fig. 17. A viable phagocyte may reënter the alveolar wall, the nucleus entering first, drawing its load of crystals, as Santa Claus does his pack.

Phagocytes not killed by their moderate load of either opaque, nonrefractive or transparent birefractive flecks are observed either within the alveolus or in the stroma of the lungs. In such phagocytes the living nucleus appears as a "red eye" and these living phagocytes encompass air borne or blood borne dust, hemoconia and other organic material that may have been deposited in the alveoli, and they transport them either along the lymph channels toward the hilum or toward the periphery of

the lung or they may congregate or aggregate in adjacent alveoli.

Such living phagocytes may reënter the wall of the alveolus, the nucleus first entering through a small chink in the alveolar wall, drawing its cell packed body through after it (Fig. 17). Many kodachrome micrographs and microscope sections from which these observations were made are, as we have said before, available for students and critics alike.

The manner in which these dust laden phagocytes congregate in small, irregularly spheroidal masses in adjacent alveoli is of great importance, but it will be considered in a section devoted specifically to the pathology of the dust nodule and the dust tubercle.

COLLAGEN VERSUS CONNECTIVE TISSUE

The dense areas in the lungs which cause roentgen shadows in cases of silicosis or pneumoconiosis have hitherto been considered to be connective tissue. But, as a matter of fact, our observations have indicated that connective tissue is singularly absent in the parenchyma and stroma of the lung, even in typical cases of silicosis. These deposits in the lung, be they (*a*) masses, (*b*) nodules, or (*c*) radiating sheaths, are caused by dust, dust laden phagocytes and collagen, either in the alveoli or in the stroma of the lung.

FIG. 18. Collagen, *not connective tissue*, is one of the principal factors in pneumoconiotic (silicotic) pathology. It is laid down in various patterns relatively free of nuclei, and it is easily differentiated from (cont. in Fig. 19)

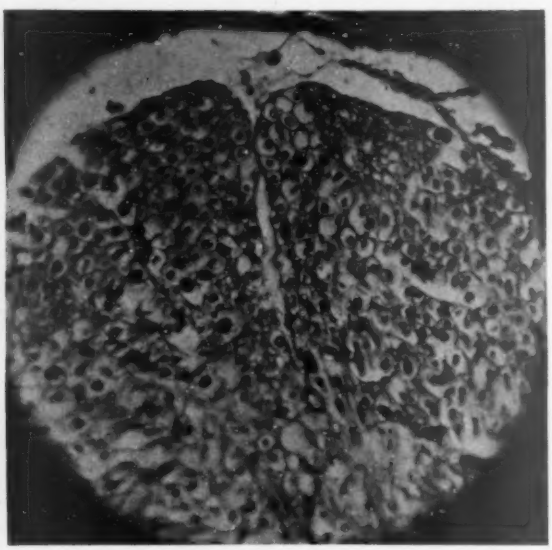

FIG. 19. Reparative connective tissue which in its early stage is characterized by cells with large, clear bodies and large nuclei; as the connective tissue ages, the cells become fusiform and eventually very long, with small oval nuclei. Collagen acts as a filler in the chinks between the cells.

Collagen in sections stained with hematoxylin and eosin is inconspicious and difficult to differentiate from other structures, particularly smooth muscle fibers, but when collagen is stained with Masson's trichrome stain, it is easily recognized and is in great contrast with the smooth muscle tissue, which is stained red. The collagen (Fig. 18) is stained blue.

Collagen as observed microscopically in cases of pneumoconiosis (silicosis) is laid down in various patterns which have been considered under four headings in the book published by the John B. Pierce Foundation.

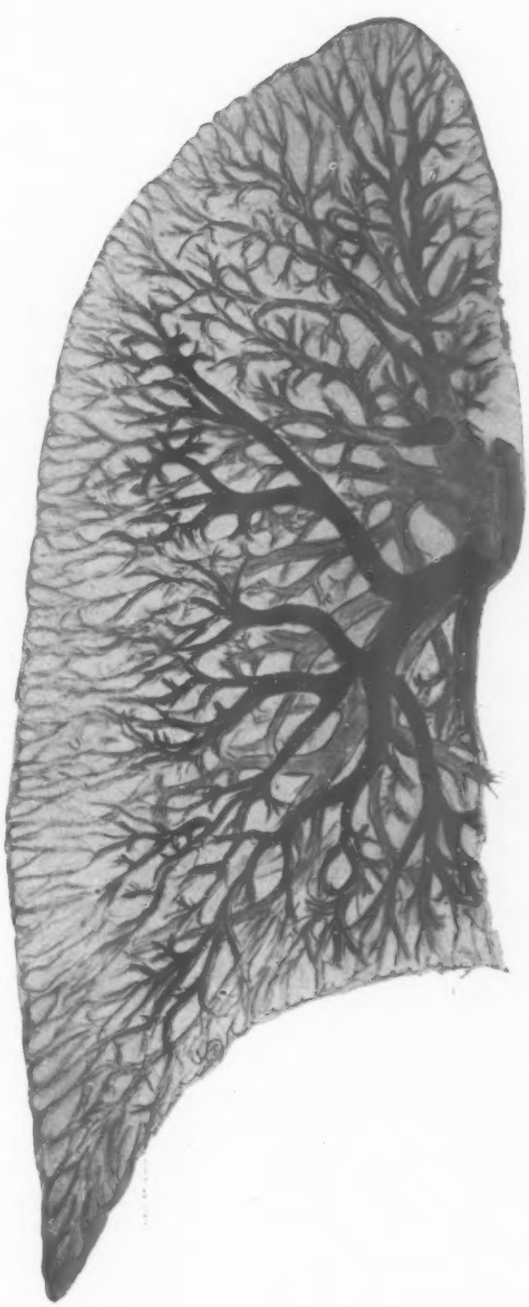

FIG. 20. Accurate tracings of the pulmonary artery as observed in a vasculogram, injected by Dr. Robb. The dark arteries supply the third lobe. The bronchi and veins run about parallel with the arteries. The blood vessel markings in the following schematic drawings are less accurate.

Six schematic drawings presenting my conception of the combined roentgenologic and pathologic findings of the six types of the disease are presented for comparison.

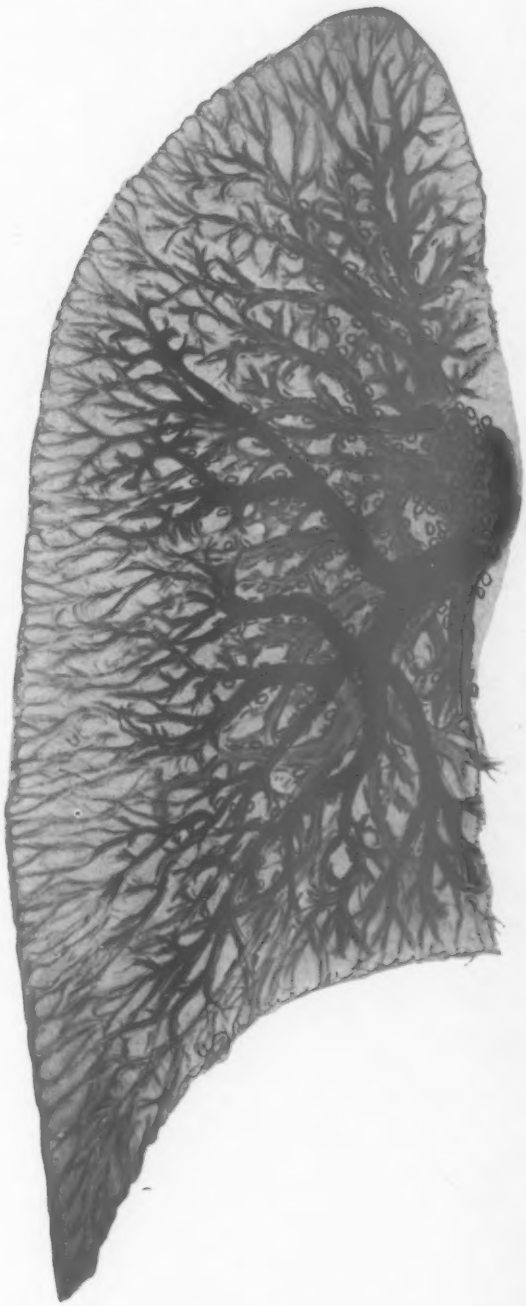

FIG. 21. *Peribronchial perivascular lymph node manifestation of Pancoast* is evidenced by accentuated hilar and linear markings that are due to dust, dust laden phagocytes and collagen deposits, laid down in laminae as sheaths surrounding the blood vessels and bronchi. The dust laden phagocytes are indicated by circles and the collagen by dashes. These findings are definite manifestations of pneumoconiosis, but often the lesion is of little clinical significance in the early stage, or even in the moderately advanced stages.

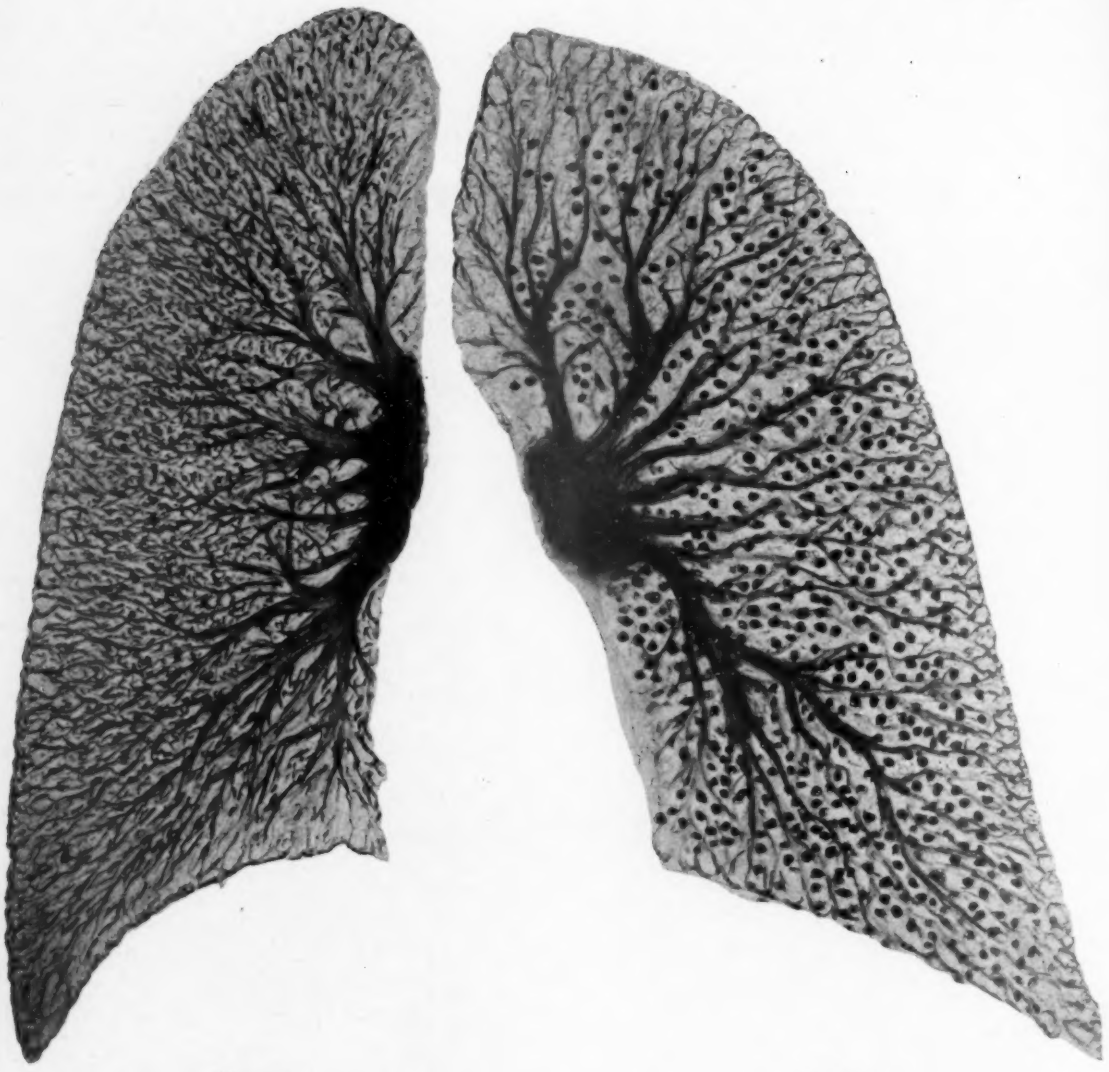

Fig. 22 Fig. 23

Fig. 22. *Vesicular type of pneumoconiosis* is evidenced by mottling with small, irregular shaped shadows that bear a definite relationship to the smaller blood vessels and bronchi, and tend to join one another. These shadows are cast by deposits of dust, dust laden phagocytes and collagen laid down as sheaths around the *smaller* blood vessels and bronchi, and in the stroma of the lung between the terminal lung lobules. These findings are not new; they were termed "M.F.U." (more fibrosis than usual) by Dr. Smith of South Africa, and as "fine nodulation" or "ground glass appearance" by others, but often they are not differentiated from the nodular type of silicosis.

Fig. 23. *Nodular silicosis* is evidenced by small, round, clear cut shadows that bear no relationship to the blood vessels and bronchi as they radiate from the hilum, and they are separated from one another by relatively well ventilated lung; these findings are readily differentiated from those observed in the vesicular type, as illustrated in Figure 22. These shadows are cast by collagen laid down in whorls, like the layers of an onion. This is the conventional type of lesion on which the diagnosis, and even the definition, of silicosis has been based.

1. Nodules or whorls, with clear cut, well defined edges; these whorls of collagen remind one of a cross section of an onion.

2. Laminae. Collagen may be laid down in laminae, as sheaths surrounding the blood vessels and bronchi.

3. Spheroidal masses or "gobs" of collagen, in irregular shapes, are laid down

largely in the deep layers of the pleura.

4. Strands, that is, fine strands of collagen, laid down in the walls of the alveoli or in the stroma surrounding the primary lung lobules.

None of these patterns of collagen is similar to any of the patterns of connective tissue. The deposition of the collagen is, I believe, a protective mechanism. Collagen is present in connective tissue but it acts as a filler between the cells. It reminds one of wax poured into a container filled with eggs; the yolks of the eggs correspond to the nuclei and the whites of the eggs to the cell bodies; the blue wax fills the chinks between the cells.

Relatively young connective tissue cells have very large nuclei and relatively clear cell bodies (Fig. 19). The spaces between these cells are filled with the blue stained collagen, which acts as a filler.

As this young connective tissue becomes older, the cells become fusiform and eventually are very greatly elongated, and the nuclei diminish in size. The waxy collagen filler between these cells is altered in shape to correspond with the aging characteristics of this connective tissue. None of the patterns of the aging reparative connective tissue is like any of the four previously mentioned patterns of collagen, as observed in pneumoconiosis or silicosis.

This finding is of more than academic interest because chronic inflammatory and tuberculous lesions of long standing develop connective tissue which contracts and deforms the thoracic cage and retracts the trachea to the side affected, whereas massive accumulations of collagen, no matter how old, do not do this. The only place in the lungs of silicotic patients in which connective tissue grows is within the lumen of the blood vessel, and this subject will be discussed later.

The normal gross and microscopic anatomy of the lung is essential, and is considered in our complete text, but space does not permit of its consideration here. A tracing of the pulmonary artery (Fig. 20) must serve as a substitute for this.

SIX TYPES OF PNEUMOCONIOSIS

To correlate the morbid changes in the lung that are manifest by dust deposits, masses of collagen, cell proliferation, air cysts, and intravascular deposits with the roentgen shadows caused by the wide va-

FIG. 24. *Tuberculoid pneumoconiosis* is evidenced by a mottling that is so similar to that caused by pulmonary lesions of the acid-fast bacillus origin, that the two cannot be differentiated from each other roentgenologically or by microscopic examination of hematoxylin and eosin stained sections. Even the distribution of the dust lesion tends to coincide with that of the acid-fast bacillus origin. However, it can be established that this lesion is caused by dust and not by the acid-fast bacillus of Koch.

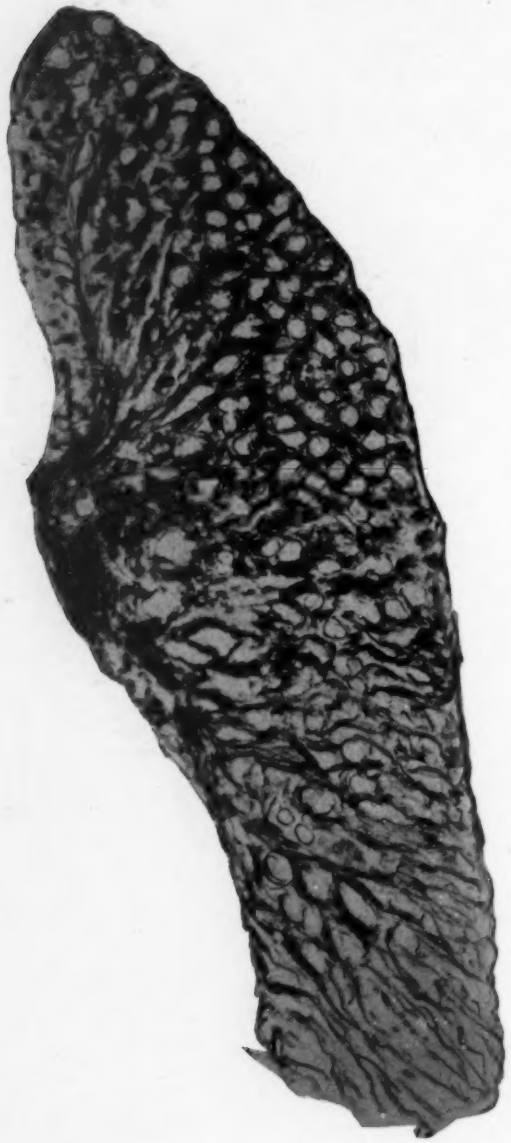

Fig. 25. *Cystic type of pneumoconiosis* (previously described in our preliminary report as "pock marking type") is shown on the original roentgenograms by black spots on a white background. This is the direct antithesis of the nodular type, which is evidenced by white spots on a black background.* These black spots are air cysts that are caused by a dilatation of the terminal lung lobules that have become dilated because of the ball-valve action of deposits of collagen or dust, which encroaches upon the bronchioles and terminal air passages and allows the air to enter, but not to exit. The walls surrounding these cysts are thickened in contradistinction to the emphysematous blebs of asthmatic origin or compensatory dilatation.

riety of findings previously illustrated, six schematic drawings based partly on roentgenologic findings and partly on pathologic findings, are presented.

I. Peribronchial perivascular lymph node pneumoconiosis of Pancoast (Fig. 21).

II. Vesicular pneumoconiosis involving the terminal blood vessels and bronchi (Fig. 22).

III. Nodular pneumoconiosis, popularized by the South African workers (Fig. 23).

IV. Tuberculoid pneumoconiosis (Fig. 24).

V. Cystic pneumoconiosis, referred to in our previous communications as "pock marking type" (Fig. 25).

VI. Parenchymal pneumoconiosis (Fig. 26), hitherto considered under the term "acute silicosis," "rapidly developing silicosis."

Each of the illustrations of these types has a complete explanation in the legend and therefore they will not be described in the text.

While these various types tend to overlap, we believe that social and economic justice will best serve employe and employer alike if they are considered as separate entities. In the manuscript of the book from which these data are extracted, these six types have been considered in great detail. The following descriptions, with a few illustrations, are only abstracts and of necessity cannot contain all of the data in support of our conception of this problem of dusty lungs.

1. *Peribronchial Perivascular Pneumoconiosis*

Terminology. Peribronchial perivascular lymph node manifestation is a term that was applied by Pancoast to a lesion of the lung caused by the inhalation of dust. This lesion is manifest roentgenologically by accentuation of the hilar and linear markings. Pancoast's broad approach to

* This drawing is reproduced as a positive and therefore the black spots on the original roentgenogram appear as white spots for more convenient comparison with the other five types.

the subject of dusty lungs is indicated by the title of his first paper, namely, "The effect of the inhalation of dust on the lungs." The question of whether this type of lesion should be considered as "silicosis" caused a cyclonic controversy resulting in the compilation of a definition tending to exclude it.

Etiology and Pathogenesis. In this type of lesion the inhaled dust that lodges within the alveoli is engulfed by phagocytes and transported through the lymph channels that run parallel with the blood vessels and bronchi, either toward the hilum or toward the pleura. Some of these dust laden phagocytes become blocked in the lymph channels at the hilum and others are dammed back into the lymph channels that run parallel with the larger and medium sized bronchi. As these phagocytes are lodged there they cause deposits of collagen (not connective tissue) to be laid down in laminae as sheaths around the larger and medium sized blood vessels and bronchi, as if they were wrapt in crinkled cellophane.

A schematic drawing (Fig. 21) based partly on roentgenologic findings and partly on pathologic findings illustrate my conception of peribronchial perivascular pneumoconiosis.

Roentgen Findings. The morbid changes just described as they are outlined against the air in the adjacent alveoli appear as accentuated hilar and linear markings (Fig. 27, 28 and 3). The increased density of the lung caused by these morbid changes increases the opacity of the mid lung field. This lesion is usually bilateral, relatively symmetrical, and is often accompanied by increased opacity at both bases and perhaps at both apices. This variation in density is best observed by squinting the eyes so that one does not see the structures and simply notes the degree of blackness of the roentgenogram. This can be accurately recorded by a photometer, as has been previously described. The increased hilar shadows may be circumscribed or they may radiate out along the blood vessels and bronchi, like the tentacles of an octopus. At

Fig. 26. *Parenchymal pneumoconiosis* (hitherto defined as "acute silicosis" or "rapidly developing silicosis") is evidenced roentgenologically by a general haze or diffuse cloudiness which obscures the normal markings of the lungs. This general haze or diffuse cloudiness observed in the roentgenograms is caused by incomplete filling or incomplete consolidation of the alveoli and air passages with dust, phagocytes and hypertrophied cells. Collagen deposits in any pattern are extremely scanty in this type of lesion, even in its terminal stage.

Socially and economically these subjects present the most serious problem since roentgenograms of this type show no nodulation, therefore the tendency is to consider such cases as not silicosis, and therefore not within the law.

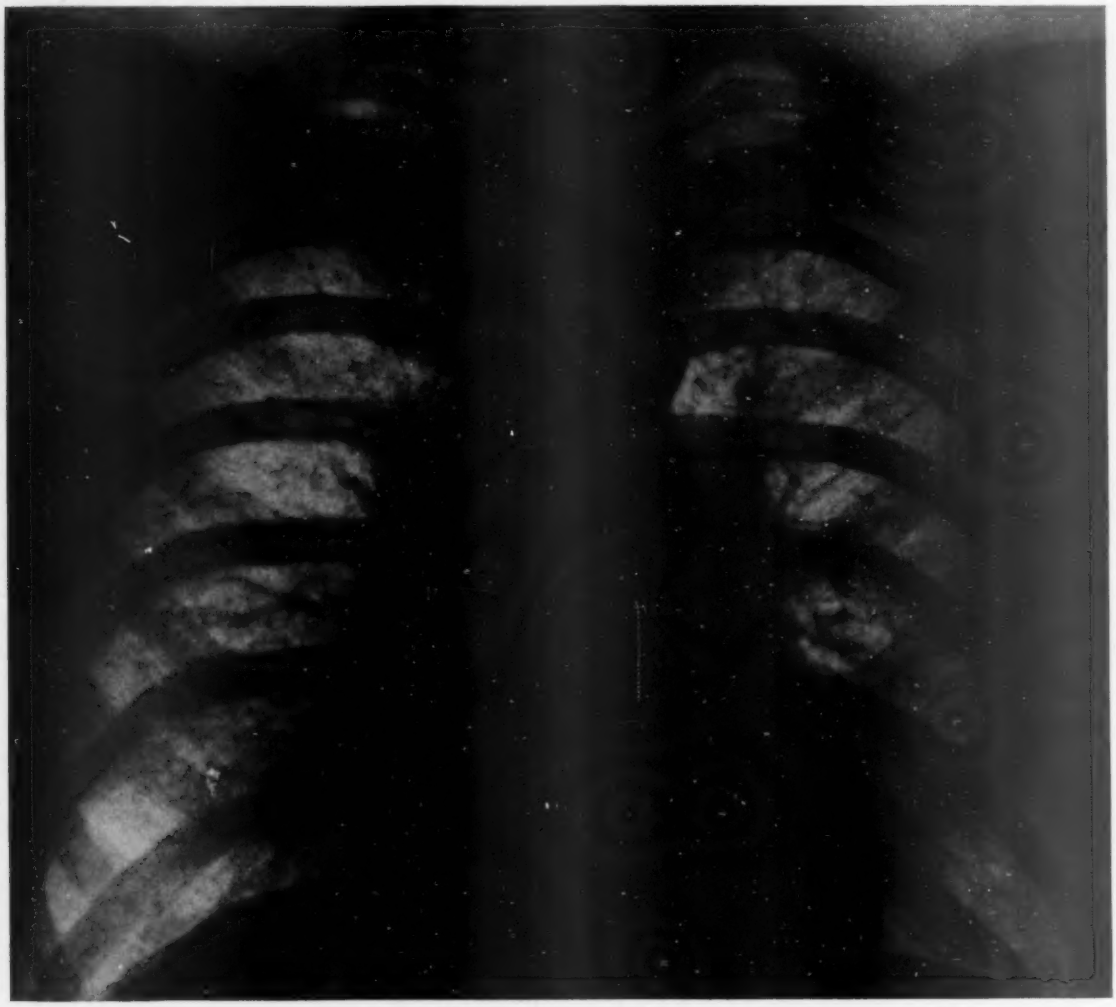

FIG. 27. (Lent by Dr. MacArthur of Noranda, Quebec.) *Peribronchial perivascular lymph node manifestation* (described by Pancoast) is evidenced roentgenologically by accentuations of the hilar and linear markings. The hilar markings may be circumscribed, or they may project out along the branches of the blood vessels and bronchi, like the tentacles of an octopus, as far as the middle and peripheral thirds of the lung. Dr. Smith of South Africa classified this particular roentgenogram as "more fibrosis than usual" and said that this lesion was found in 30 per cent of the men not working underground. A better example of this type of lesion is shown in Figure 3. These are definite manifestations of pneumoconiosis but often they are of little significance in the moderately advanced stages.

points where these increased linear markings overlap, their shadows on the roentgenogram are increased, giving a mottled appearance.

Gross Pathology. The mass at the hilum radiating out along the bronchi and blood vessels may be very dense and on gross section of the lung it may be so filled with grit that it is almost impossible to cut it with a sharp autopsy knife. These pathologic findings are best observed in large microsections that extend all the way from the hilum to the periphery of the lung, especially when they are stained with Masson's trichrome stain and used as a lantern slide.

The increased density at the hilum and around the bronchi is not due to connective tissue, and certainly not to reparative connective tissue with aging characteristics. When these structures are viewed mi-

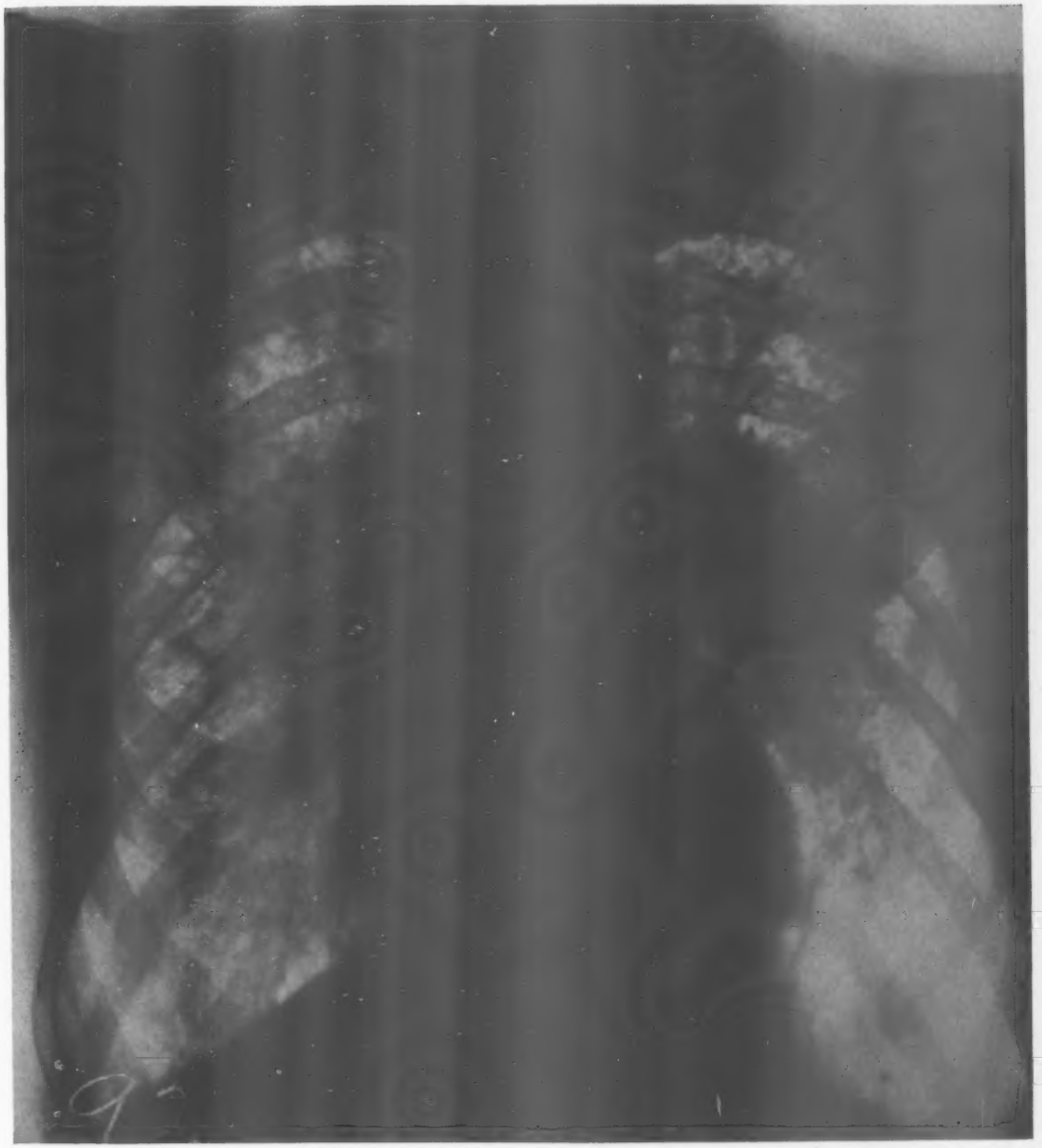

Fig. 28. In the late stages of the peribronchial perivascular lymph node manifestations, after quantities of dust have been phagocyted out of the alveolus and deposited at the hilum, around the *larger* blood vessels and bronchi, definite roentgen shadows are cast as shown in the right lung. Even this hilum, which is less involved than the left, was almost impossible to cut through with a sharp autopsy knife when sectioned for microscopic study. Socially and economically this class of lesion becomes a problem, first, because it constitutes such a large percentage of pneumoconiotic cases, and second, because it is a menace both to labor and to industry. The man who is perfectly able to work and wants to work may not be able to get a job because he has such a lesion. On the other hand, industry may be saddled with the compensation of a man with such a lesion, who is perfectly able to work, but does not want to work.

Grit at the hilum, when (cont. in Fig. 29)

croscopically they are observed to be composed of dust, both opaque non-refractive and transparent birefractive, dust laden phagocytes, collagen laid down in whorls or strands, and moderate cell proliferation. When the larger blood vessels and bronchi are cut transversely or obliquely, the sheath of dust laden phagocytes and collagen appears as a ring adjacent to the muscular coat (Fig. 30). The opaque flecks are observed in the conventional light field, and the transparent birefractive flecks of silica and the silicates, not seen in the light field, are observed when the field is rendered dark by cross polarization (Fig. 29).

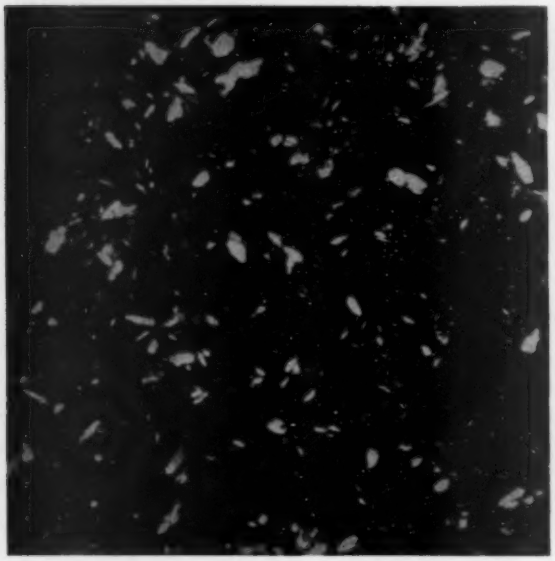

Fig. 29. Viewed with the field rendered dark by cross polarization, the huge quantities of silica crystals show why it was so dense and hard to cut.

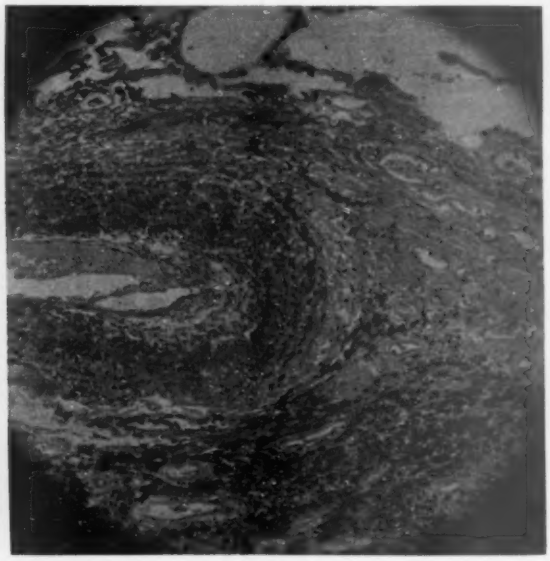

Fig. 30. Collagen above the edge of the blood vessel and dust laden phagocytes below form a sheath around an artery that is two or three times as thick as the arterial wall.

The vast majority of pneumoconiotic lesions are of this type, which is of less clinical significance than the other types of pneumoconiosis. Nonetheless, these are dust deposits and morbid changes incident to the inhalation of dust. This type of pneumoconiosis develops where there is a moderate amount of dust with a low percentage of silica or silicates. In our collection the most characteristic roentgenograms of this type occur in buffers and grinders, in miners in upper Maryland and lower Pennsylvania (loaned to us by Drs. Derr and Bridgers), and in the anthracite miners (loaned by Drs. Corcoran and Gordon of Scranton). Dr. Smith, of South Africa, whom the Canadian Government brought to their country to aid them in the solution of their silicotic problems, said that this type of lesion occurs in 30 per cent of the men not underground (but presumably inhaling some dust).

These cases rarely develop symptoms until the latter decades of life. An occasional case, with very slight roentgen findings and no dyspnea, will claim compensation. This procedure reacts unfavorably to his fellow workers, therefore these cases become potential social, economic and industrial problems. However, these problems must be faced and solved, if possible.

There is just as much injustice on the part of the employer or management who may refuse to employ a man because of

roentgen findings observed in pre-employment examination.

II. *Vesicular Pneumoconiosis*

Terminology. This term is not altogether satisfactory, but it is used as a suggestion, hoping that someone will offer a better one. Although this lesion is closely associated with the peribronchial perivascular lymph node manifestation just described, it is such an important factor in the problem of dusty lungs that we are loath to subjugate it to the term "peribronchial perivascular lymph node manifestation" which is under a cloud of disapproval since it was eliminated from the diagnostic, social and economic problems by the definition of silicosis hitherto discussed.

Etiology and Pathogenesis. The etiology and pathogenesis of the vesicular type of pneumoconiosis are identical with the previous type, except that the dust laden phagocytes and small strands of collagen are laid down in the lymph channels surrounding the *smaller* blood vessels, bronchioles and terminal lung lobules. These dust deposits around the terminal lung lobules, especially if they are black, are easily visible as a ring, many of which when joined together give a peculiar net-like appearance to the surface of the lung just beneath the pleura. This vesicular lesion is usually associated with the peribronchial perivascular type just described, but it may predominate to such an extent that it detracts attention from the involvement of the larger and medium sized bronchi and blood vessels. My conception of this lesion is illustrated in the schematic drawing (Fig. 22) previously presented in comparison with the other five types.

Roentgen Findings. The roentgen findings of the vesicular type of pneumoconiosis are evidenced by a bilateral, relatively symmetrical, diffuse mottling with a pattern not so clear cut and well defined as a silicotic nodule. These small roentgen shadows are irregular in shape, often like a Y or sections of a jig-saw puzzle. Prongs of one such shadow seem continuous with those of another. These small irregular shadows bear a definite relationship to the shadows cast by the smaller blood vessels and bronchi, which is the direct antithesis to the silicotic nodules which are separated from each other and *do not* bear a definite relationship to the blood vessels and bronchi. These roentgen findings are by no means new; they have been described by roentgenologists but frequently have not been differentiated from the nodular type of pneumoconiosis.

Roentgenologists have used such terms as "fine nodulation," "ground glass appearance," "M.F.U." (more fibrosis than usual) and "anteprimary," etc., to designate them. Smith of South Africa designated these as "M.F.U. +, ++, +++." In their more advanced stages he designated them as "anteprimary +, ++, +++." These terms he applied to distinguish this lesion from the nodular type of silicosis to which the term "primary silicosis" was applied. These roentgen findings have usually been considered as early stages of nodular silicosis, but we believe that these lesions do not develop into typical nodular type of silicosis described in Type III. We believe that this lesion should be considered as a pathologic entity, perhaps associated with the mid lung peribronchial perivascular lesion, of which, as a matter of fact, it forms a part.

The gross appearance of these lungs differs very little from the normal and certainly very little from the peribronchial perivascular lymph node manifestations. The morbid changes are best illustrated in large microsections stained with Masson's trichrome stain projected onto the screen as a lantern slide.

In the mid lung field of such a section one observes a moderate sized artery and bronchus cut transversely (Fig. 32). These structures are surrounded by a sheath of dust laden phagocytes and laminae of collagen which appear as rings similar to that described and illustrated in Type I but they are smaller. A small branch of this medium sized artery cut longitudinally gives the key

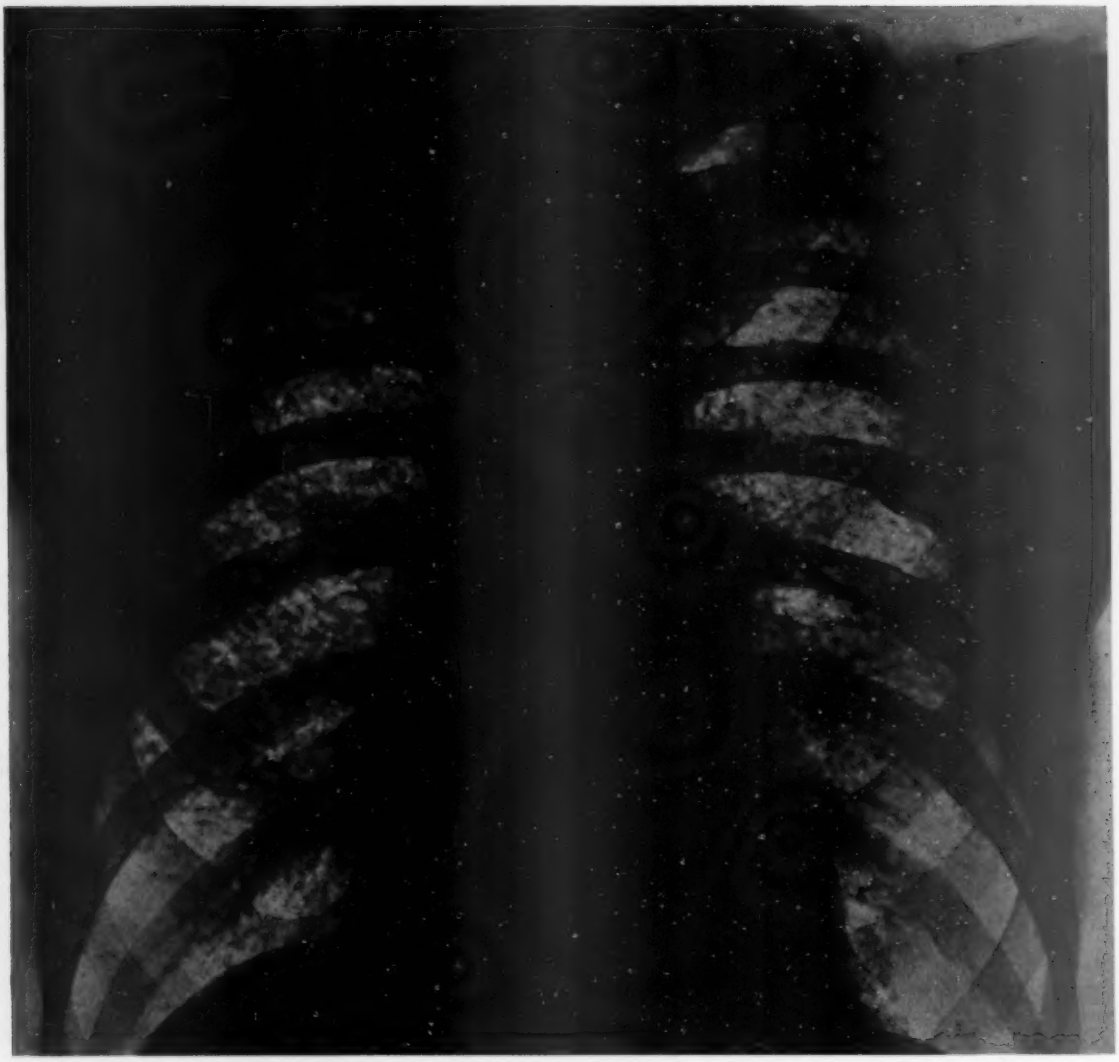

FIG. 31. (Lent by Dr. Gordon, Scranton, Pa.) *Vesicular Type of Pneumoconiosis. Roentgen Findings.* Bilateral, symmetrical mottling of small, irregular spots that are joined together but do not have clear cut and well defined edges. These irregular spots bear a definite relationship to the terminal blood vessels and bronchi. These findings are not new; Dr. Smith of South Africa referred to them as "M.F.U.+, ++, and +++," in their earlier stages, and as "anteprimary silicosis+, ++, +++," in their more advanced stages, thus distinguishing them from the nodular, which he considered "primary silicosis." These small, irregular spots, characteristic of the vesicular type of pneumoconiosis are caused by (cont. in Fig. 32)

for correlating the vesicular type of pneumoconiosis with the mid lung lesion described by Pancoast and illustrated in Figures 3 and 27.

This terminal blood vessel cut longitudinally shows the artery for its entire length (Fig. 32). The dust sheath which surrounds it appears as parallel black lines that lie adjacent to the outer surface of the muscular coat. This black sheath corresponds almost identically with the characteristic mottling observed roentgenologically; even the bulging is similar to the beading observed in the roentgenograms. The fact that these thick walled blood vessels and bronchi cast shadows on the roentgenogram that are so definitely associated with the pattern of the normal lung differentiates

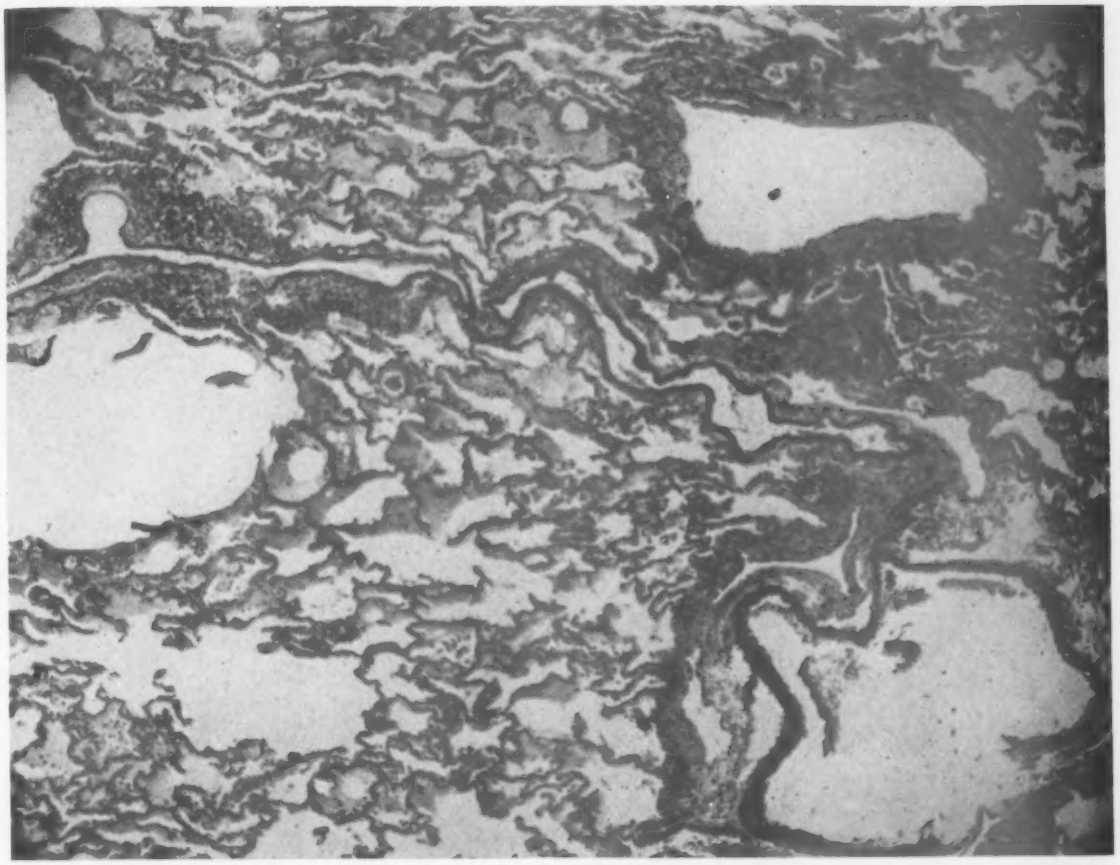

Fig. 32. Deposits of dust laden phagocytes and collagen laid down in sheaths around the smaller blood vessels and bronchi; even the bulging or budding at the left of the illustration is similar to the roentgen findings. Dilated lung lobules are seen to the left, and a medium sized artery and bronchus cut transversely, with sheaths of collagen and dust, are seen to the right.

them from the nodular whorls (silicotic nodules) which are denser, rounder and bear no relationship to the blood vessels and bronchi. Compare Figures 22 and 23 of the schematic drawings and Figures 31 and 33 of the roentgenograms for the similarity and differentiation of these two types of lesions.

The terminal lung lobules supplied by these partially constricted or contracted or compressed bronchioles or air passages tend to become distended (Fig. 32) and in this respect they somewhat overlap the type of pneumoconiosis to which we have applied the term "cystic." These cases will therefore be discussed in greater detail under that heading. I believe that this type of lesion is of greater clinical, social and economic significance than the peribronchial perivascular type involving the larger and medium sized bronchi. I have seen one case of this type with extreme dyspnea, far out of proportion to the density of the lung. The alveoli were markedly dilated. There was clubbing of the fingers with deformity of the nails due to interference with the pulmonary circulation.

III. *Nodular Pneumoconiosis (Silicosis)*

Terminology. The term "nodular silicosis" has become so firmly fixed in the medical literature that it may seem audacious even to limit its application to the specific lesion it was originally intended to describe. This term was given the stamp of approval by the Committee which drafted the defini-

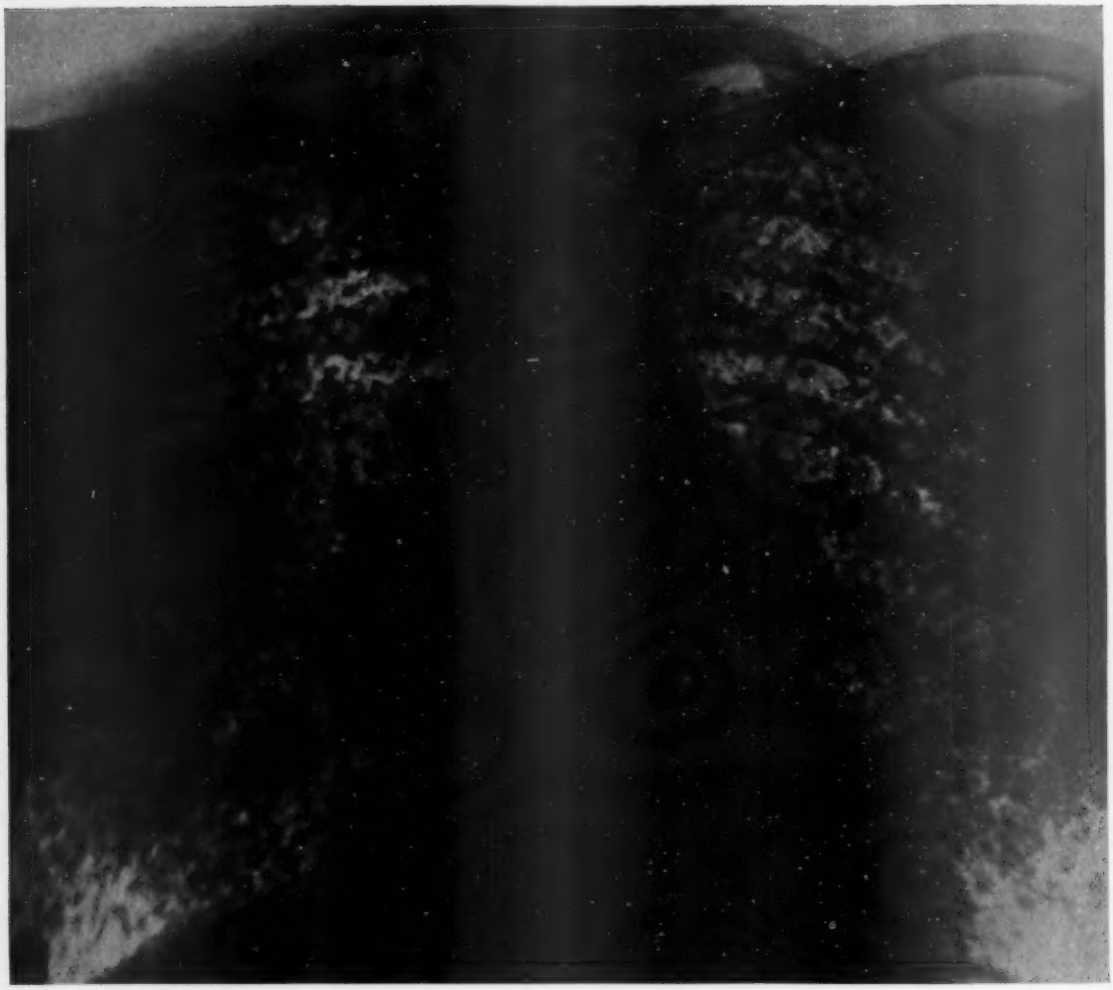

Fig. 33. (Lent by Dr. Klein, Perth Amboy, N. J.) *Nodular Pneumoconiosis. Roentgen Findings.* Bilateral, symmetrical mottling, with small round spots having clear cut edges, that bear no relationship to the radiating blood vessels and bronchi; in the earlier stage they are separated from one another by well ventilated lung. There they may coalesce, especially in the right lower mid lung field where the three lobes overlap, producing the "pawnbroker's sign."

Clinical Manifestations. Although these lungs are shot full of nodules, the patient was free from symptoms; the lesion was found accidentally while he was being examined for fractured ribs. Absence of symptoms in this type of lesion is the rule rather than the exception.

Socially and economically many of these subjects are able to work at hard labor. Should they be prevented from getting a job or losing the one they have? Legislative acts are primarily built around this type of lesion hitherto considered silicosis, in contradistinction to the two previous types. The small, round, clear cut spots, characteristic of this type of lesion, are caused by (cont. in Fig. 34)

tion of silicosis published in the *U.S. Public Health Bulletin*, in August, 1935. No better term could have been found for these small, isolated, spherical masses in the lung, and therefore in this communication we are limiting the term "nodular" to the specific lesion it was specifically intended to describe. This type of lesion was originally described, or at least popularized at the turn of the century, by the South African workers. Whether they intended to limit this term to nodular whorls (or silicotic nodules), which they considered to be due to fibrous nodules of connective tissue, is not

clear. But the definition published in the *Public Health Bulletin* is fairly specific, in limiting silicosis to this type of lesion. There is some doubt in my mind if those who formulated the definition differentiated the nodular type of silicosis from the vesicular type, on the one hand, or the chronic tuberculoid type, on the other.

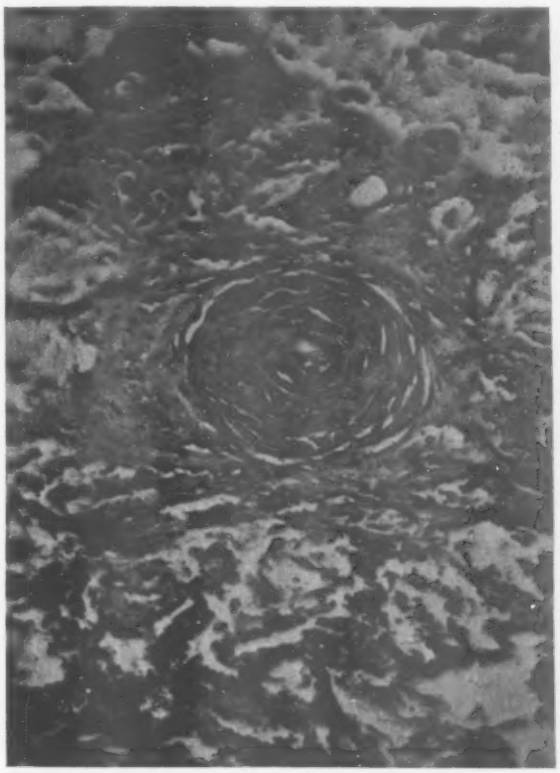

Fig. 34. Whorls of collagen laid down like the layers of an onion. These small spherical masses which are clear cut in outline have been termed "silicotic fibrous nodules," but they are not composed of connective tissue.

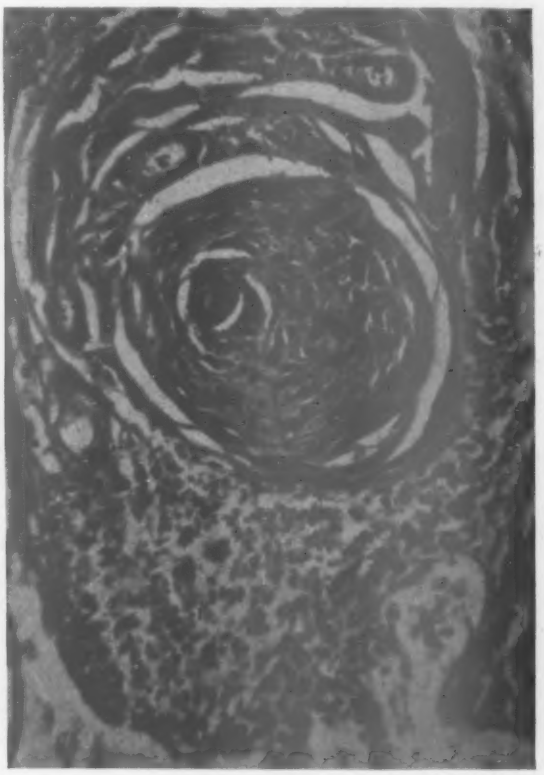

Fig. 35. These nodules are supposed to develop in lymph follicles, but such follicles are not only *not* the home of the nodule, but they are freer of collagen than any other structure of the lung. When whorls do develop near such a follicle it pushes the follicle aside.

Etiology and Pathogenesis. The etiology and pathogenesis of the nodular whorls to which the term "silicotic nodule" has hitherto been applied are not as clear as the etiological and pathological factors of the other five types. These nodules are not connective tissue nodules; certainly they are not composed of reparative connective tissue with aging characteristics of the cell. They are composed of collagen laid down like the layers of an onion (Fig. 34), and adult nodules contain very little dust, either opaque nonrefractive or transparent birefractive flecks within the interstices of these layers of collagen. In what we believe to be the nascent nodule, that is, the young early developing nodule, there are more deposits of dust and dust laden phagocytes and less collagen than in the adult nodule. This evidence seems to indicate that the nodular whorl of collagen (so-called silicotic nodule) constitutes a protective mechanism in which clumps of dust laden phagocytes are incarcerated within the interstices of these layers of collagen.

If this theory is true, the question of what happens to the dust within the phagocyte as the whorl becomes of age is prob-

lematic. It has hitherto been stated and rather universally accepted that lymph follicles throughout the lung are the location of the silicotic nodules or collagen whorls. Our evidence definitely tends to prove that these lymph follicles are not only *not* the home of the nodule, but that nodules are less likely to occur in the lymphatic follicles than in any other histologic structure of the whole lung. Indeed, when a nodular whorl develops adjacent to a follicle, it pushes the follicle out of the way instead of growing into it, indicating that the two are incompatible (Fig. 35).

A schematic drawing compared with the others helps to convey my conception of this type and to differentiate it from the other types.

Roentgen Findings. The term "nodulation" or "roentgen-ray nodulation" has hitherto been rather loosely applied to shadows cast by pathologic structures other than the silicotic nodule or collagen whorls. If we were to speak of bilateral symmetrical mottling rather than nodulation, and if one looks for the points of differentiation between the different types of mottling, it becomes evident that there is a difference between the clear cut, well defined round or oval shadows that are bilateral, relatively symmetrical and bear no relationship to the blood vessels and bronchi (Fig. 33) and the irregular shaped mottling described under the term "vesicular pneumoconiosis" (Fig. 31), and that both of these should be differentiated from the pock marking type, characterized by black spots on a white background, as observed in the original roentgenogram. These black spots on a white background are negative shadows that are cast by accumulations of air in the cystic type of pneumoconiosis and will be considered under the cystic type.

Microscopic Examination. Viewed microscopically two or more whorls of collagen may be connected to form conglomerate nodules, and groups of nodules may be connected by strands of collagen to form small localized areas of dense lung. Viewed with higher power, the nodule is observed to be composed of layers of collagen that are relatively free of nuclei (Fig. 34). It has clear cut, well defined edges and is relatively free of dust, either opaque or transparent birefractive flecks. When the nodules are viewed with the microscope field rendered dark by cross polarization, they are often singularly free of silica, compared with the surrounding structures.

Social and Economic Problems. This type of lesion is slow in developing, perhaps because the nodules form a protective mechanism. The patients are often singularly free of symptoms, even when the lungs are filled with these nodular whorls of collagen (Fig. 33) (silicotic nodules).

IV. *Chronic Tuberculoid Pneumoconiosis*

Terminology. Tuberculoid pneumoconiosis is a lesion that looks like pulmonary tuberculosis but is not caused by the acid-fast bacilli of Koch. Certain other terms herein used are defined as follows:

Dust Nodule: This term is applied to small, irregularly spherical masses that are relatively free of collagen, but are composed of opaque, non-refractive and transparent, birefractive flecks of dust, that usually have a definite relationship to the peripheral blood vessels and bronchi.

Silicotic Nodule: The term "silicotic nodule" is applied to whorls of collagen widely disseminated throughout the lungs, bilateral, relatively symmetrical, that bear no constant relationship to the blood vessels and bronchi.

Dust Tubercle: This term, in contradistinction to the dust nodule on the one hand, and the silicotic nodule, on the other, is applied to irregularly spherical masses composed of collagen combined with dust laden phagocytes that is so similar in appearance to the pulmonary tubercle of acid-fast bacillus origin that the two can scarcely be differentiated by microscopic examination of hematoxylin and eosin stained sections, but special stained sections show that the dust tubercle contains no acid-fast organisms.

Acid-Fast Pulmonary Tubercle: This

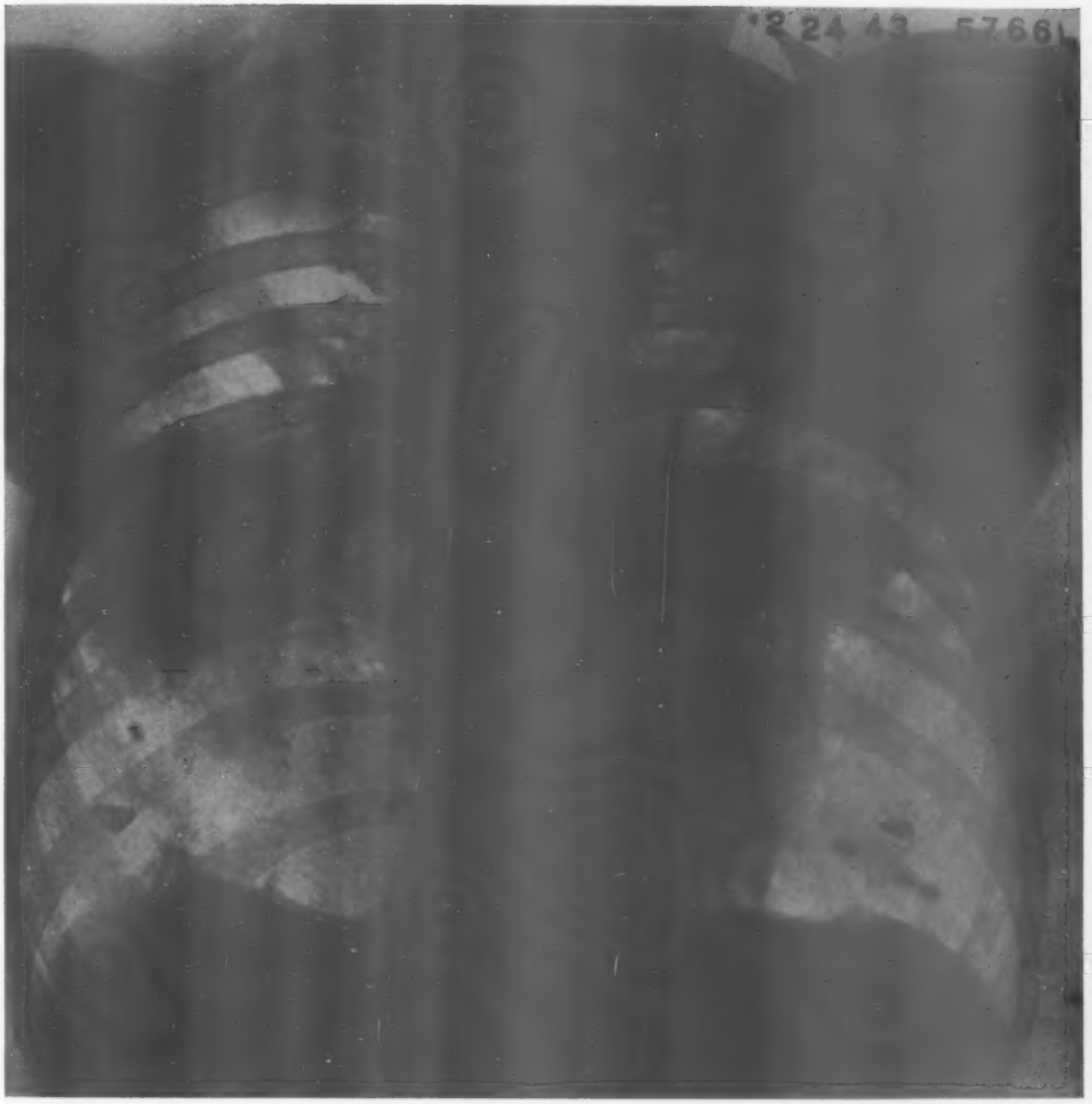

Fig. 36

term is reserved for pulmonary tubercles that can be proved to be caused by the acid-fast bacilli of Koch.

This tuberculoid lesion has not hitherto been differentiated from nodular silicosis, on the one hand, and silicotuberculosis, on the other.

Etiology and Pathogenesis. Reverting to the anatomy and histology of the lung and to the travelogue of the phagocyte hitherto recorded, it becomes evident that the phagocytes emerge from the alveolar wall and engulf foreign body flecks. These phagocytes may not all progress through the lymphatic channels toward the hilum or toward the pleura. In the tuberculoid type of pneumoconiosis these dust laden phagocytes congregate in an alveolus or in adjacent alveoli and together with collagen form small, irregularly spherical masses that correspond in size, shape and structure to the pulmonary tubercles of acid-fast bacillus origin.

A schematic drawing of this type (Fig.

FIG. 37

24) aids one in differentiating it from the other five types, but it does not aid in its differentiation from pulmonary tuberculosis of acid-fast bacillus origin.

Roentgen Findings. The outstanding characteristic of the roentgen findings (Fig. 36) is that they so closely simulate findings observed in pulmonary tuberculosis of acid-fast origin that the experienced roentgenologist, specialist in tuberculosis, and even experts in the problem of pneumoconiosis and silicotuberculosis, would unhesitatingly state that the patient was suffering from pulmonary tuberculosis. They might qualify this statement by saying that if there were an authentic history of adequate exposure to silica, the case might be considered as silicotuberculosis. This lesion is usually bilateral, is by no means symmetrical, and often there are large, dense areas in one or the other apex, with areas of infiltration of various sized nodules or tubercles in the mid lung field of the opposite lung. These tubercles or nodules bear no relation-

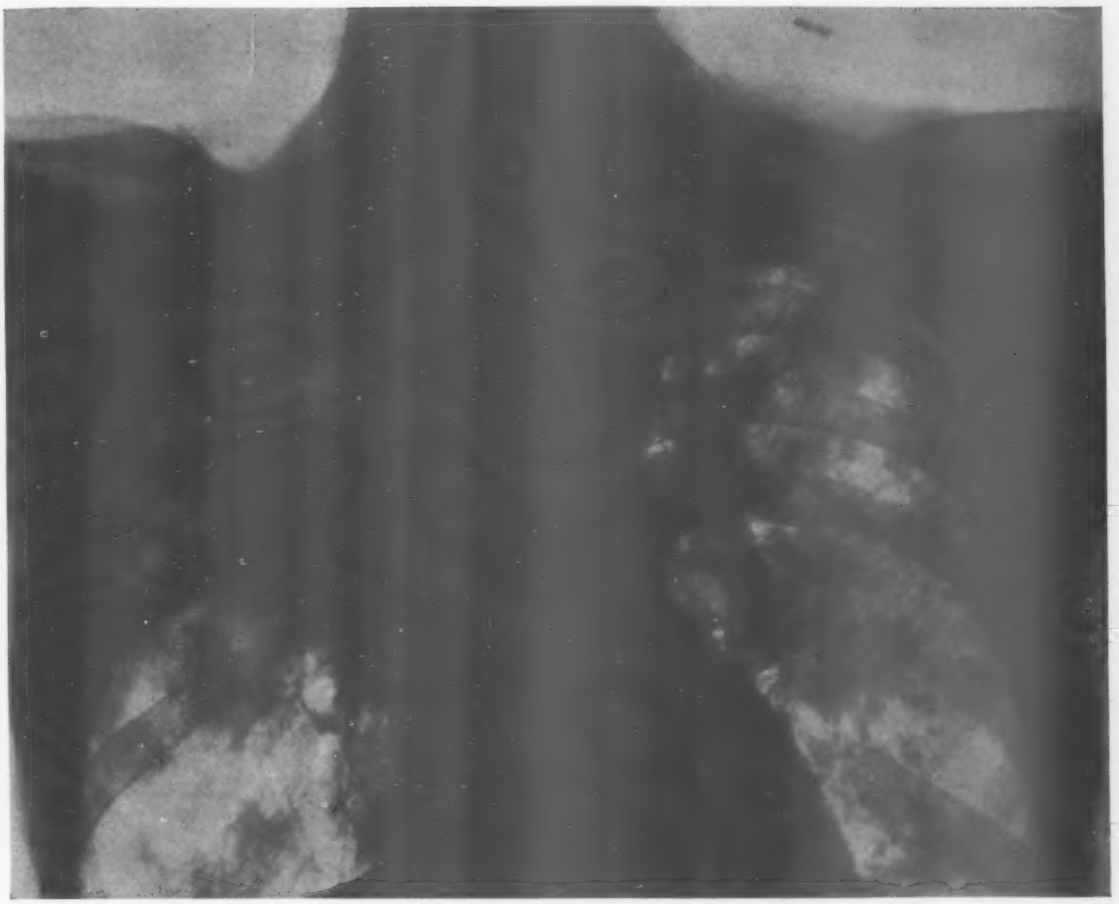

Fig. 38

Fig. 36,* 37†, 38‡. *Tuberculoid Pneumoconiosis.* This is a lesion which resembles pulmonary tuberculosis but is not caused by the acid-fast bacillus of Koch.

Figures 36, 37 and 38 are illustrations shown over this continuous caption. One of these is a lesion caused by dust without acid-fast bacilli of Koch; another is caused by the acid-fast bacilli of Koch, without dust; and the other occurred in a man with a thirty-two year history of intensive exposure to dust in a mine, but he now has acid-fast bacilli in his sputum. To emphasize the similarity of these lesions and the difficulty of differentiating them, they are reproduced for comparison with one another, without at this time prevealing which is which.

* Referred by Dr. H. J. Noering, Valatie, N. Y.
† Referred by Dr. Harold Gartner, Valhalla, N. Y.
‡ Lent by Dr. William Corcoran, Scranton, Pa.

ship to the normal radiating linear markings.

Points of Differentiation. The roentgen findings that tend to differentiate tuberculoid pneumoconiosis of dust origin from the pulmonary tuberculosis of acid-fast bacillus origin are as follows:

1. With the dust lesion, the consolidated areas are homogeneous, even in roentgenograms made with the Potter-Bucky diaphragm.

2. The lesion lies closer to the periphery of the lung.

3. The heart shadow is rendered indistinct along its left border.

4. The trachea is only slightly deviated or retracted, if any.

5. The thoracic cage is symmetrical,

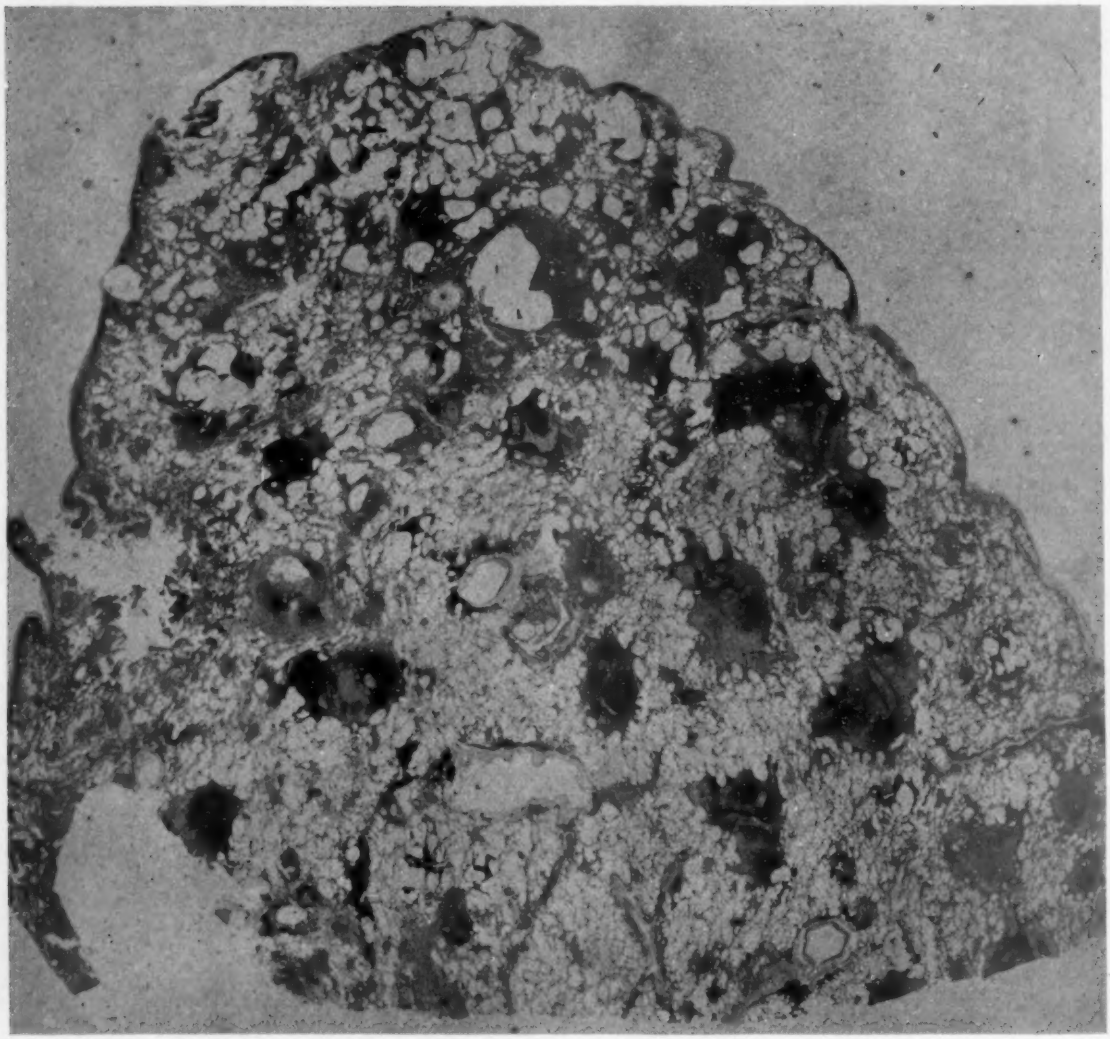

Fig. 39

large, and barrel shaped; the ribs are not drawn together, as is often the case in chronic fibroid tuberculosis.

A gross specimen, cut longitudinally, shows large and small dense areas widely disseminated throughout the lung.

Macroscopic examinations of large microscope sections, somewhat magnified and preferably thrown on the screen as a lantern slide, show the distribution of the tubercles of various sizes and shapes that are almost identical with the size, shape and distribution of tubercles of acid-fast bacillus origin. Two large microscope sections (Fig. 39 and 41), one of dust origin and the other of pulmonary tuberculosis of acid-fast bacillus origin, are included to show the great similarity between the tubercles of dust origin and those of acid-fast bacillus origin. Between these two illustrations we have inserted an illustration by Delafield (Fig. 40) in which he made an etching on stone showing pulmonary tubercles as observed with low power magnification. In our illustrations we have not designated which of the two (Fig. 39 or 41) is of acid-fast bacillus origin and which is of dust origin. For the time being we have left that to the reader in order to impress him with the similarity of the lesions.

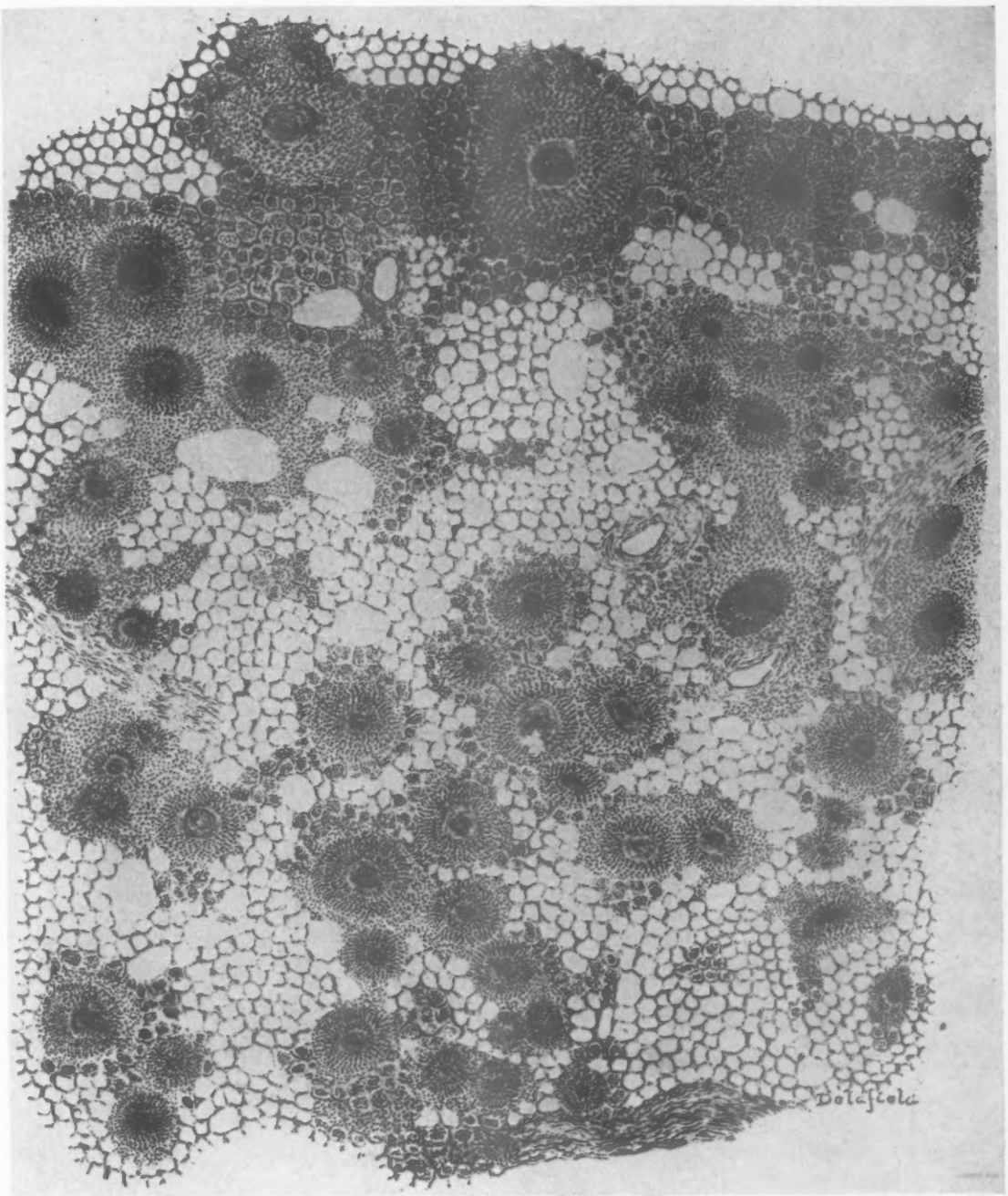

Fig. 40

Two medium power photomicrographs, one of an acid-fast bacillus tubercle and the other a dust tubercle, are also shown, but likewise, in proof of their similarity it is not revealed at the present time which is which.

Problem of Differentiation. A large microscope section, well stained with hematoxylin and eosin, was presented to a high ranking pathologist interested in this subject.

He expressed the opinion that the lesion was silicotuberculosis. When it was suggested to him that it was silicosis and

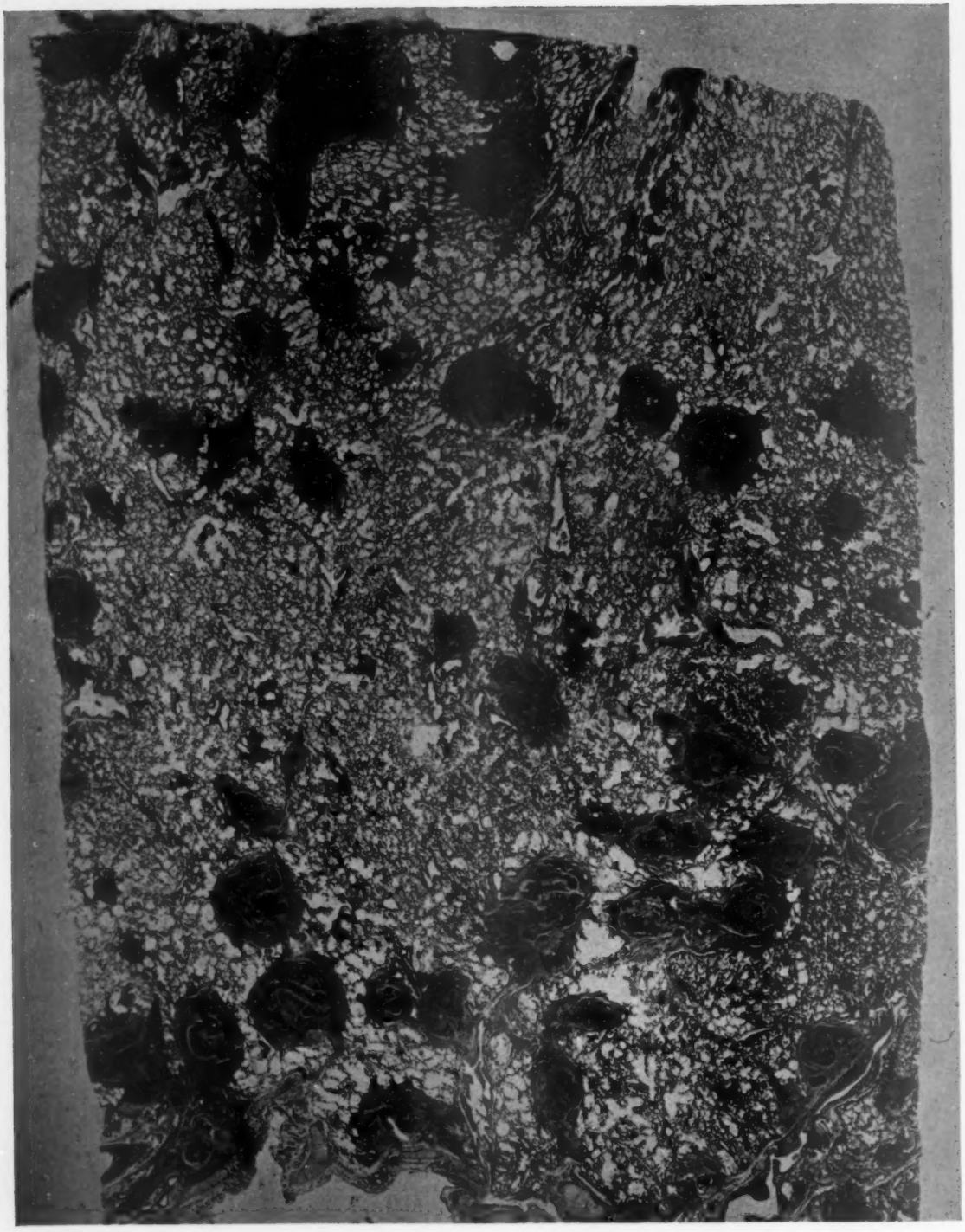

FIG. 41

FIG. 39, 40 and 41. Small irregular spherical masses, separated from one another by relatively normal lung. One of these is a reproduction of a drawing made by Dr. Francis Delafield which he personally etched on stone to illustrate the findings which he observed in pulmonary tuberculosis. This drawing of pulmonary tubercles was made before the discovery of the acid-fast bacillus was announced by Koch in 1882; one of the other two lesions is caused by dust without acid-fast bacilli, and the other by acid-fast bacilli. To emphasize the similarity of these dust and acid-fast bacillus tubercles, we are not now revealing which is which.

not silicotuberculosis, he reiterated his statement: "I mean silicotuberculosis." However, we believe that we have incontrovertible evidence obtained by special microscopic procedures and special stains to eliminate the possibility of this lesion having been caused by the acid-fast bacillus of Koch, and definite evidence that it is caused by dust, largely silica, of granite origin.

Three chapters in our book are devoted to the similarity between dust lesions and those of acid-fast bacillus origin, etiology and pathogenesis of both, and methods and criteria in proof of the fact that the tuberculoid lesion is of dust origin and not of acid-fast bacillus origin. In this article, it is impossible to present that evidence. We believe that this type of lesion is of more serious social, economic, industrial and legislative significance than are any of the previously described types.

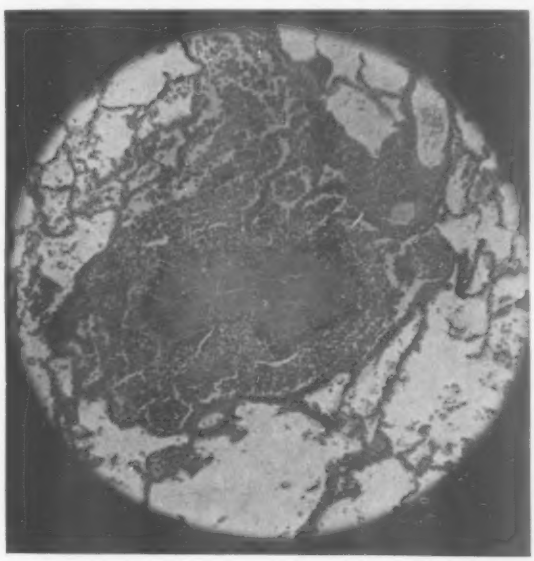

Fig. 42

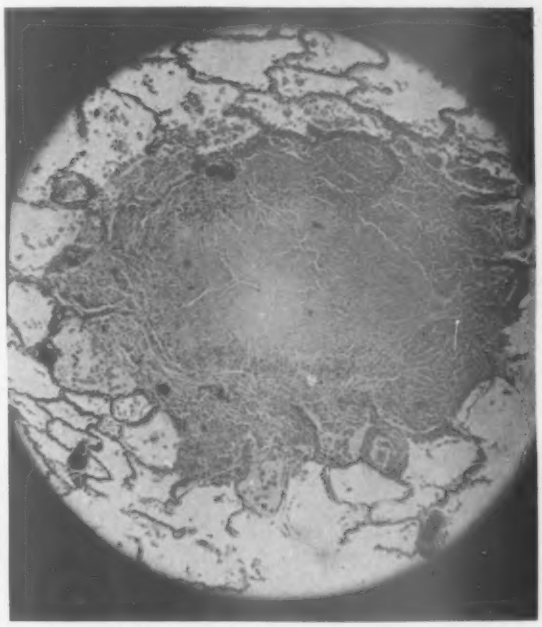

Fig. 43

Fig. 42 and 43. Even when these small, spherical masses, called tubercles, are studied microscopically with the conventional light field after being stained with hematoxylin and eosin, those that are caused by the acid-fast bacilli cannot be distinguished from those caused by silica of granite origin. More than sixty years ago Delafield described such types of pulmonary tubercles, but at that time the cause of pulmonary tubercles was unknown; the acid-fast bacillus of Koch had not yet been discovered.

The most classical of the seven types was the amorphous tubercle, the center of which was so disintegrated that its structures could not be recognized and therefore it was termed "amorphous." Surrounding this central part was a rim of viable cells. This type of tubercle is well illustrated in Figures 42 and 43, one of which is of dust origin and the other of acid-fast bacillus origin. Which is which?

v. *Cystic Pneumoconiosis*

Terminology. The term "pock marking" was applied to this lesion in our preliminary report on dusty lungs, but the term "cystic" is more scientific and more applicable. Cystic pneumoconiosis is evidenced by air cysts which are dilated pulmonary lung lobules. The bronchioles or air passages leading to the terminal lung lobule become occluded or compressed by masses of collagen or dust laden phagocytes which surround them or impinge upon them. This deposit of collagen and dust causes a valve-like action which allows air to enter the terminal lung lobule but does not allow its exit.

The schematic drawing (Fig. 25) is made in black and white to coincide with the other five types and even then it does not show the lesion as well as if it were pre-

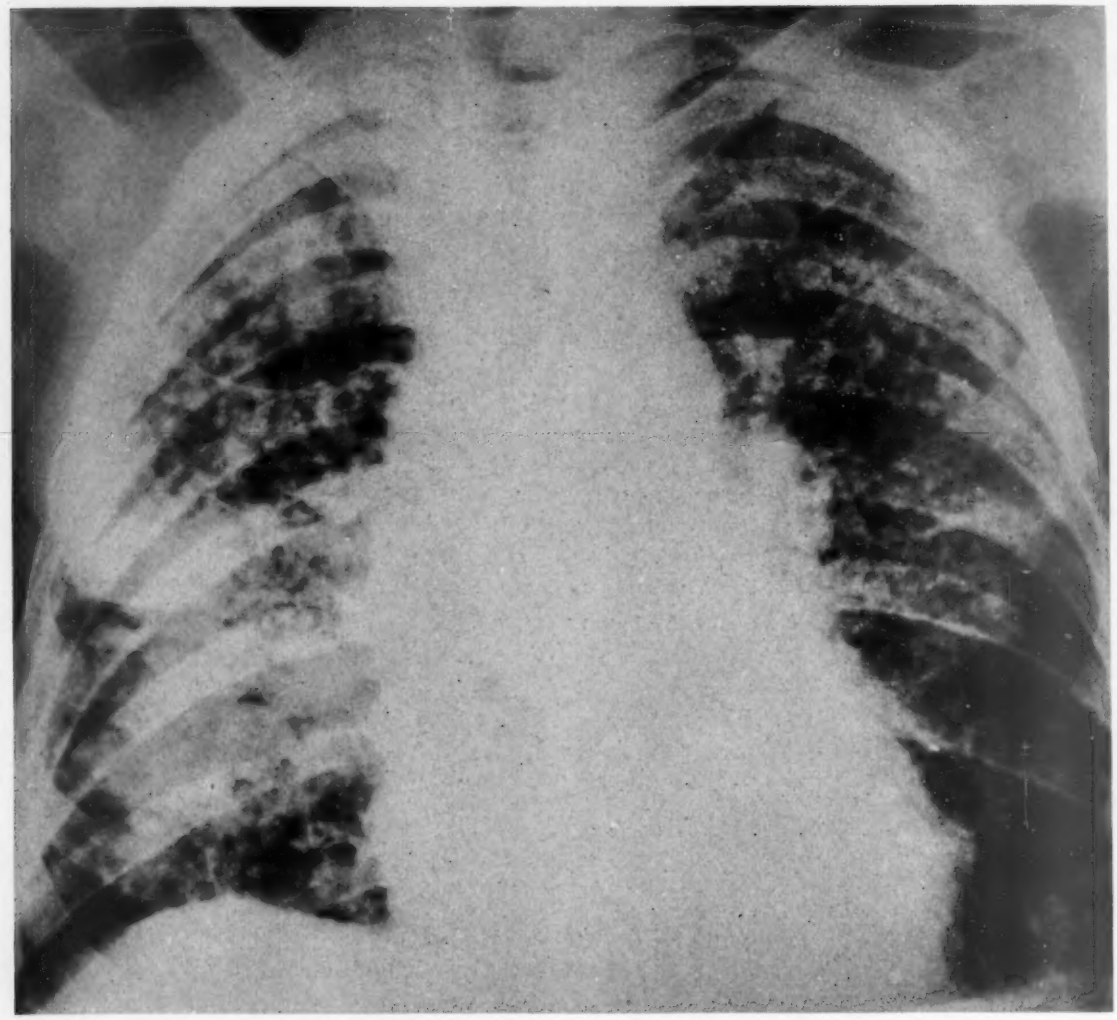

FIG. 44. *Cystic Pneumoconiosis* ("pock marking"). *Roentgen Findings.* Bilateral but not symmetrical mottling evidenced by small black spots on a white background, or surrounded by a white ring, is best shown in the second and third interspaces. This illustration is reproduced as a negative because the black spots on a white background are more readily seen than white spots on a black background. The black spots are air cysts caused by dilated terminal lung lobules; air is inhaled but its exit is blocked by the ball-valve action of collagen and dust deposits. Quantities of dust and collagen deposits are laid down in these lungs, some in the form of definite whorls, but in the original roentgenograms the black spots within white areas predominate. Socially and economically these constitute a much more serious problem than any of the previous types, except possibly the late stages of tuberculoid type.

sented as a negative drawing. This is done because it is much easier to see a black object on a white background, and for this reason all other roentgenograms are reproduced as positives rather than negatives, because it is the black or dense objects which constitute the roentgen findings in which we are interested.

Roentgen Findings. These spherical cysts that contain air within relatively solidified portions of the lung are surrounded by rings of dense tissue, cast black spots on a white background (Fig. 44), the direct antithesis of the roentgen appearance of the nodular type of pneumoconiosis (Fig. 33), which is manifest by white spots on a black background in the original roentgenograms. These lesions are bilateral, often are not as

symmetrical as the first three types, and while they do contain white spots caused by nodular whorls of collagen or deposits of dust, yet the cystic appearance predominates.

Gross Specimen. On the cut section of the gross specimen, these cysts appear as black spots located largely in its peripheral third of the lung.

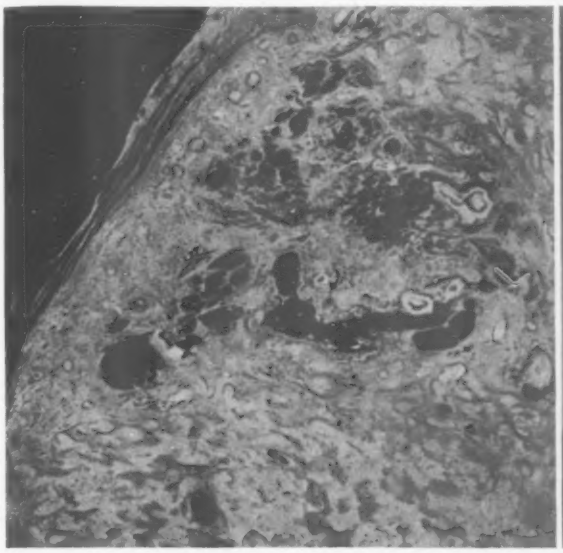

Fig. 45. A negative print of a microscopic section which is comparable with the roentgen findings; that is it shows black spots on a white background. It reminds one of a slice of Swiss cheese. Large and small cysts are seen, one of which is shown microscopically in Figure 46.

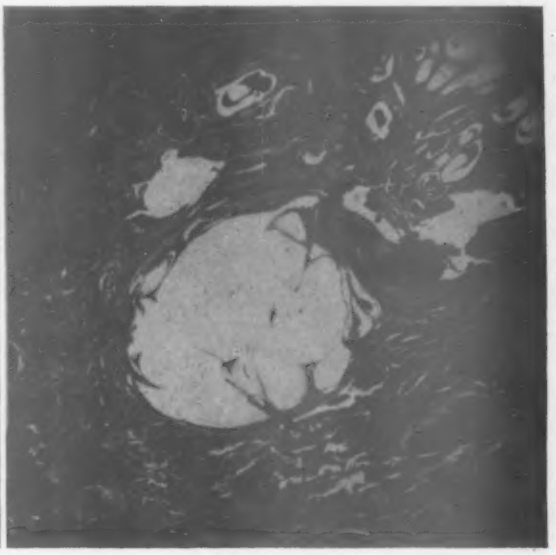

Fig. 46. This shows how the air compresses the partition within the terminal lung lobules. Adjacent alveoli surrounding the air cysts are compressed and deformed. These are to be differentiated from emphysematous blebs.

Magnified photomicrographs of large microscope sections show these air cysts as round black spots in a white area (Fig. 45) that remind one of a slice of Swiss cheese; the black spots correspond to the holes in the cheese. A comparison of a negative print of a large microscope section (Fig. 45) with the roentgenogram (Fig. 44) emphasizes the great similarity of the pathologic and the roentgenologic findings.

When air cysts are observed in less dense regions of the lung, they may be surrounded by rings of collagen and these morphologic rings cast ring-like shadows in the roentgenograms. When these air cysts are viewed microscopically with low power magnification, the partition forming the alveolar wall within the lung lobule is compressed and as the air cyst dilates it, it likewise compresses adjacent alveoli that are displaced peripherally (Fig. 46). These air cysts are not to be confused with emphysematous blebs, although the mechanism that causes it is similar to the *modus operandi* of the emphysema of asthma except that in asthma the lesion is functional whereas in the cystic pneumoconiosis it is organic. Likewise, the dyspnea of asthma is intermittent or periodic, whereas the dyspnea of this cystic type is constant.

Social and Economic Problems. This type of lesion constitutes a more serious social and economic problem than any of the previous types, except possibly the advanced stages of the tuberculoid type.

Less distinct cysts that are caused by terminal lung lobules that are not definitely dilated but are somewhat surrounded by strands of collagen, as described in the vesicular type, are somewhat similar to these air cysts, but the terminal lobule is not so

much dilated and the rings surrounding it are less distinct than in the cystic type. Such terminal lung lobules filled with air but without thick walls are observed in Figure 32 which illustrates the dust sheath around the terminal blood vessels and bronchioles. This manifestation of the lesion is recognized in many roentgenograms if one looks for it. However, dust, dust laden phagocytes and collagen are not the only morbid changes which may cause these roentgen findings, and therefore air cysts themselves are not pathognomonic of pneumoconiosis, but they are very suggestive.

VI. *Parenchymal Pneumoconiosis (acute silicosis, so-called acute silicosis, or rapidly developing silicosis)*

(*A*) *Acute Pneumonic Parenchymal Pneumoconiosis and* (*B*) *Acute Tuberculoid Pneumoconiosis.*

Terminology. Parenchymal pneumoconiosis has hitherto been termed "acute silicosis," "so-called acute silicosis," or "rapidly developing silicosis." This type of pneumoconiosis (silicosis) was first described by MacDonald, Piggot and Glider, and then by Chapman of the Massachusetts General Hospital, with the roentgenograms lent to me by Dr. George W. Holmes. This lesion was later discussed by Gardner under the term "so-called acute silicosis." It seems wiser to us to discuss this lesion under the two headings of this section, viz., acute pneumonic parenchymal pneumoconiosis and acute tuberculoid pneumoconiosis, as being comparable with the acute pneumonic type of pulmonary tuberculosis or acute phthisis, in contradistinction to acute general miliary tuberculosis where the tubercles are small and separated from one another by relatively healthy lung tissue.

Definition. Acute pneumonic parenchymal pneumoconiosis is a lesion manifest by incomplete filling of the alveoli with dust, dust laden phagocytes, cholesterol, the clefts of which are observed in the microsection, and cells desquamated from thickened alveolar walls. Certain large areas in the lung are rendered almost completely avascular whereas adjacent portions of the lung are hypervascular; at least the capillaries are immensely dilated, containing many times the normal content of blood. This type of lesion is singularly free from nodular whorls of collagen (silicotic nodules) and there is relatively little collagen of any pattern in any portion of the section except in the walls of the alveoli where there are fine strands that have a tendency to occlude the pulmonary capillary circulation.

A schematic drawing (Fig. 26), fully described, is presented adjacent to the other five types to illustrate the characteristic roentgenologic and pathologic findings.

Roentgen Findings. The roentgen findings are manifest by a diffuse general haze or cloudiness, involving both lung fields (Fig. 47). This lesion is bilateral but usually not symmetrical. Within these areas where there is a general haze, there are localized patches of increased density that suggest more advanced degrees of consolidation. These lungs are singularly free of clear cut, well defined nodules on which the diagnosis of silicosis is usually based, and unless one is familiar with this type of lesion he would scarcely even think of this being silicosis.

Such was the experience of those who encountered the Gauley Bridge group of cases, most of which were of this type.

Macroscopic Examination. The magnified microsections serve to correlate the roentgenologic and pathologic findings, and when a large section stained with Masson's trichrome stain is thrown on the screen, collagen is conspicuously absent (Fig. 48). A low power microscopic examination of large sections demonstrates the scarcity of collagen whorls (silicotic nodules) and a very slight amount of collagen that is present in any form. In the section stained with trichrome, the areas which are hypervascular stand out in brilliant red contrast to the avascular areas in which one can find scarcely a single red blood cell when the section is examined with higher power.

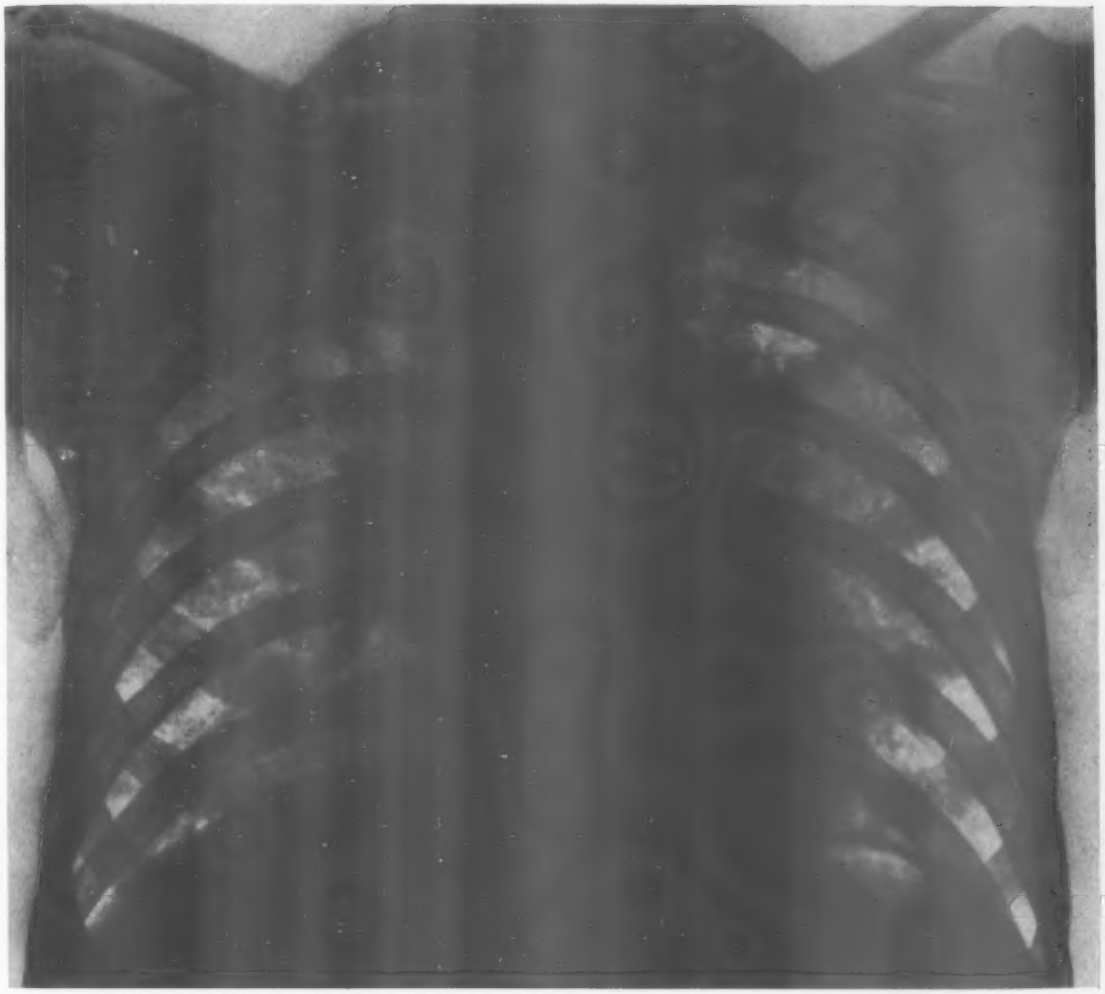

Fig. 47. (Lent by Dr. Harless, Gauley Bridge, W. Va.) *Parenchymal Pneumoconiosis* (acute silicosis or rapidly developing silicosis). *Roentgen Findings.* This is shown by a general haziness, without characteristic nodulation, and without accentuation of the linear markings. These findings are bilateral but not symmetrical. Owing to the absence of nodulation or accentuated linear markings, lesions of this type may not be recognized by the roentgenologist unless he is familiar with it.

Clinical and Occupational History. Male, aged about twenty-two; had twenty months' intensive exposure to dust with a high silica content. He was dead within twenty months from his first exposure.

Socially and economically, these subjects present the most serious problem and in the past injustice has been done to this group of men because the subjects were unfortunate in that they did not have the well developed nodulation on which the diagnosis and definition of the disease are based. The indefinite haziness in these cases is caused by (cont. in Fig. 48)

Microscopic Examination. The walls of the alveoli in the avascular areas are markedly thickened; the cells are hypertrophied and many are proliferated and desquamated. The alveoli contain quantities of dust, dust laden phagocytes composed of both the opaque nonrefractive and transparent birefractive dust flecks. These dust laden phagocytes are also observed in the stroma of the lungs. Fine strands of collagen laid down in the walls of the alveoli occlude the capillary circulation, and as we suggested in our first article on "The dyspnea of silicosis, what causes

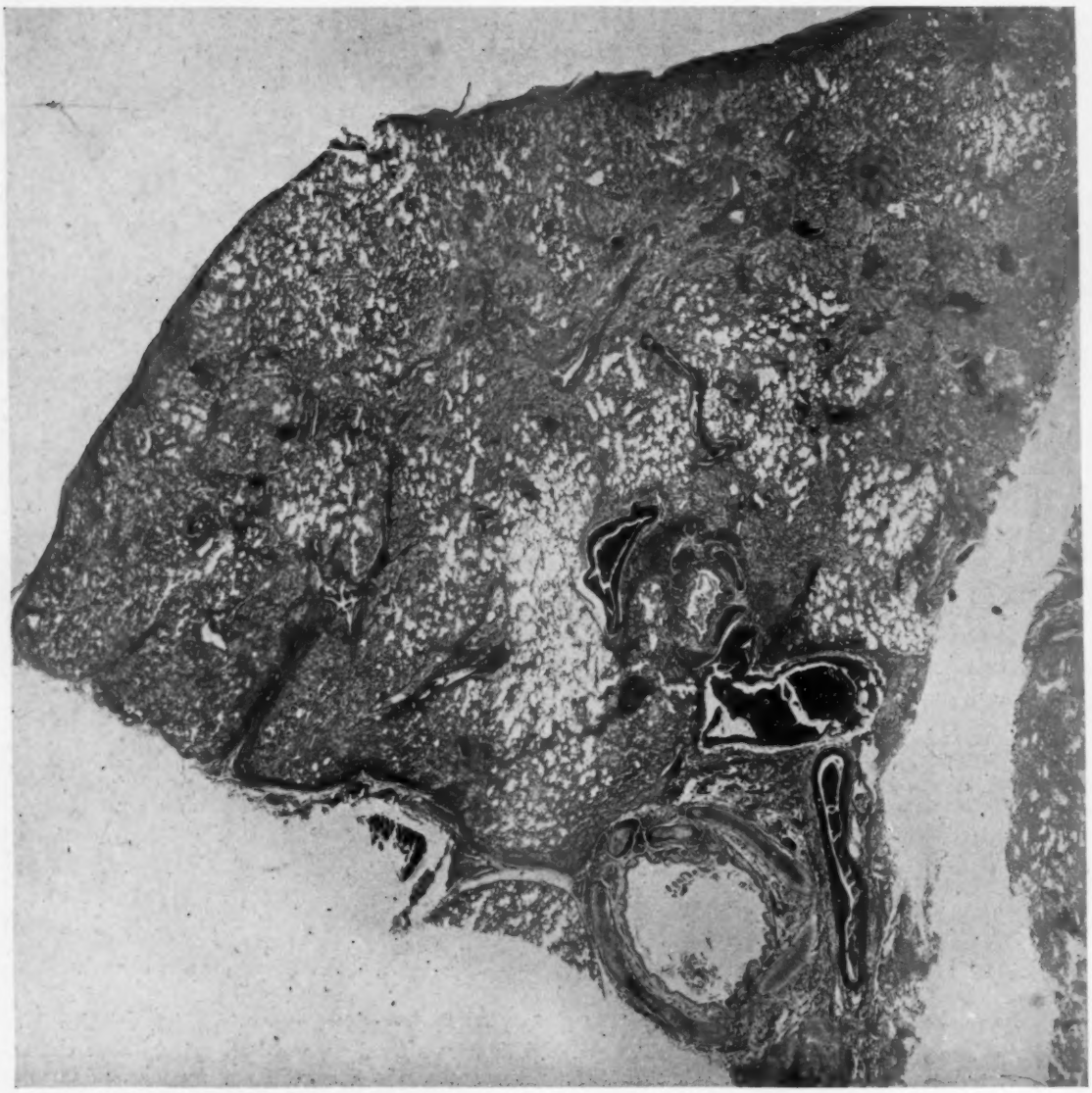

Fig. 48. Incomplete consolidation or filling of the alveoli and terminal air passages of the lung with dust, dust laden phagocytes and hypertrophied, desquamated cells from the walls of the alveoli. Collagen in any pattern is singularly scanty. This statement is hard to prove in this reproduction in black and white, but the absence of collagen in the original trichrome stained section is most convincing. Some portions of the microscopic section are avascular, whereas adjacent regions are hypervascular, as is elaborately described and illustrated in the full manuscript of our book, from which this is taken.

it?" we believe that this capillary occlusion is one of the causes, perhaps the principal one. In the hypervascular areas which lie adjacent to the avascular areas, the walls of the alveoli are thinned, the cells are not hypertrophied or proliferated, and these alveoli contain relatively little dust, either opaque or transparent, birefractive.

Acute tuberculoid pneumoconiosis is a lesion similar to that of the acute pneumonic manifestation (acute silicosis) in that the lesion is composed of dust, dust laden phagocytes and desquamated cells deposited in the alveoli and terminal air passages. Like the pneumonic lesion, collagen whorls (silicotic nodules) or collagen in any pattern are singularly scanty except

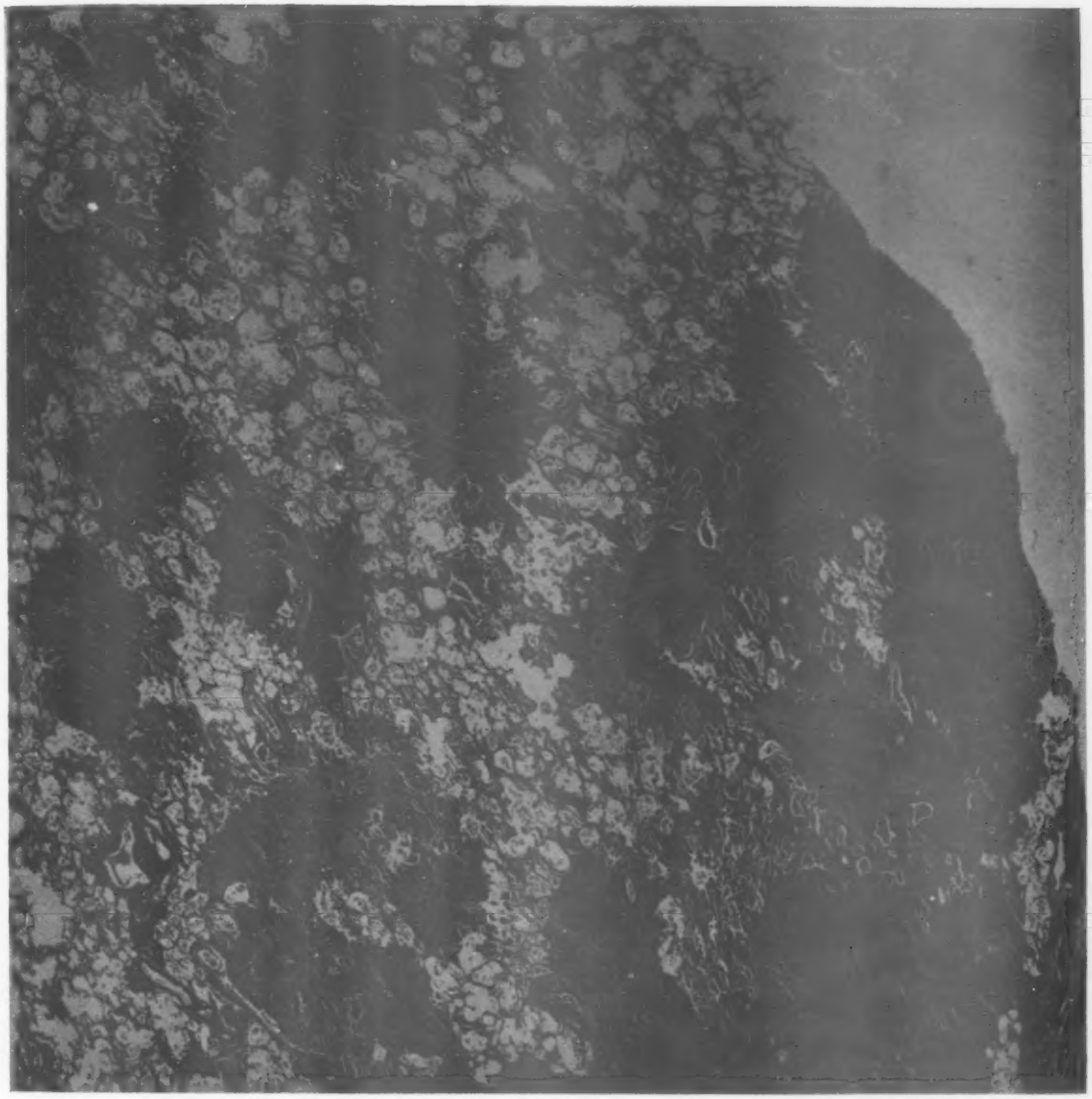

Fig. 49

perhaps as fine strands of collagen in the walls of the alveoli.

Acute tuberculoid pneumoconiosis is similar to the *chronic tuberculoid type* in that its distribution is the same, namely, the dust laden phagocytes congregate or aggregate in small, irregularly spherical masses that are similar in size, shape and distribution to those of acute miliary tubercles of acid-fast bacillus origin. Indeed, the lesion is so similar to that of acid-fast bacillus origin that we are using two low power photomicrographs (Fig. 49 and 50); one is a lesion of dust origin and the other of acid-fast bacillus origin, and to impress upon the reader the similarity of these lesions, we shall not at this time reveal which is which.

A similar field viewed with low magnification and by the conventional light field method shows a mass of phagocytes clumped together to form a tubercle (Fig. 51). No opaque dust is observed in these phagocytes, even by high power magnification, but when the field is rendered dark by cross polarization, thousands of transparent

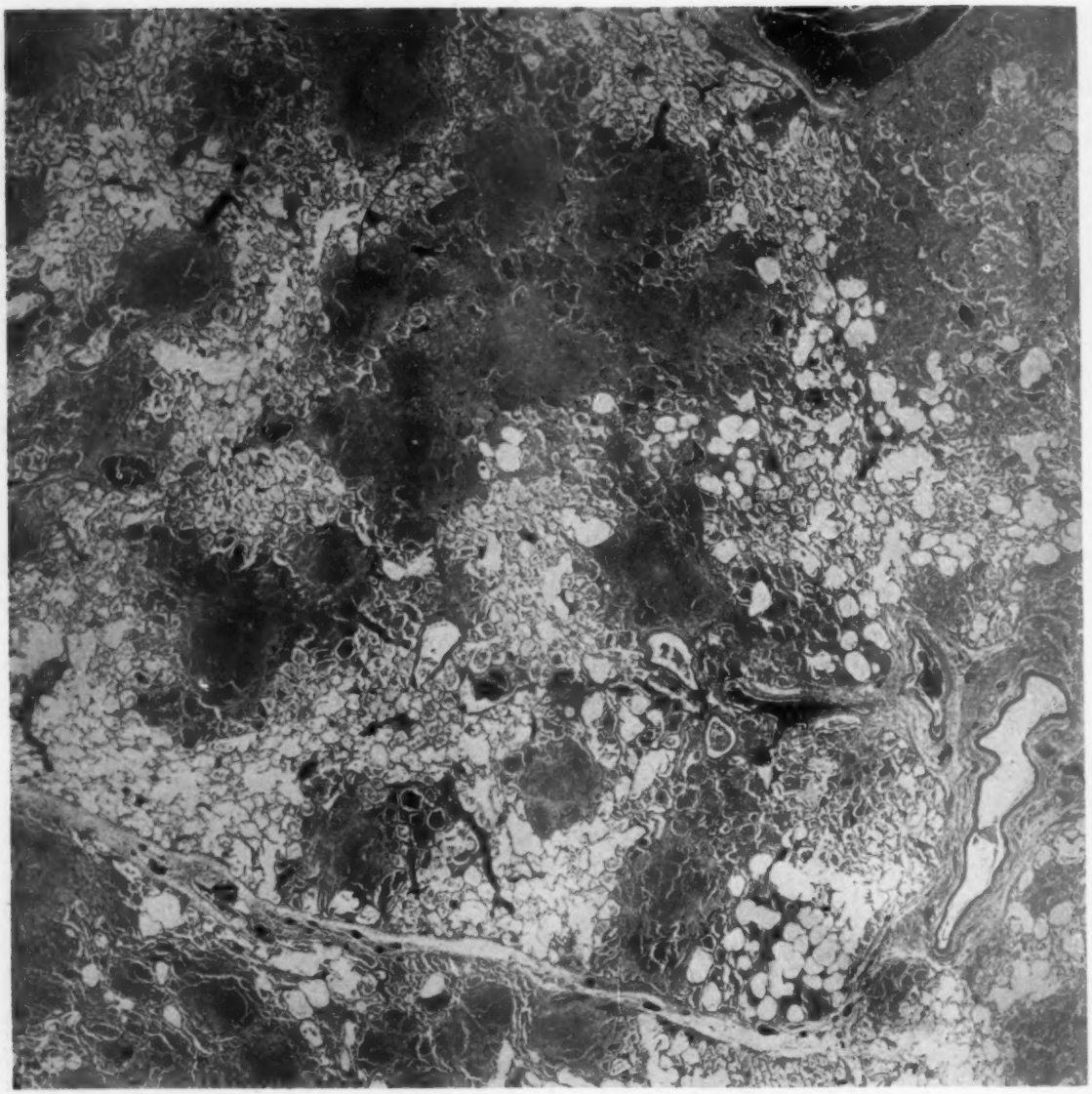

FIG. 50

FIG. 49 and 50. Acute pulmonary tuberculosis of acid-fast bacillus origin and acute pulmonary tubercles of dust origin are so similar that they cannot be differentiated from each other by microscopic examination of hematoxylin and eosin stained sections with the conventional light field method of examination. Microscopic examinations of these sections stained for the acid-fast bacilli showed that the pulmonary tubercles in one were literally loaded with acid-fast bacilli, whereas the pulmonary tubercle in the other case was singularly free of acid-fast bacilli. We are not yet stating which is which.

flecks become startlingly brilliant (Fig. 52). They are even impressive as they appear as white spots in a dark field, as reproduced in the halftone cut, but this figure is far from satisfactory to illustrate the startling appearance of these fields as observed in the original microscopic examination, or even as shown in kodachrome photomicrographs used as lantern slides or film slides.

Illustrations of chronic tuberculoid pneumoconiosis that were originally inserted in this text under the section devoted to that subject are now inserted just after the illustrations of acute tuberculoid pneumo-

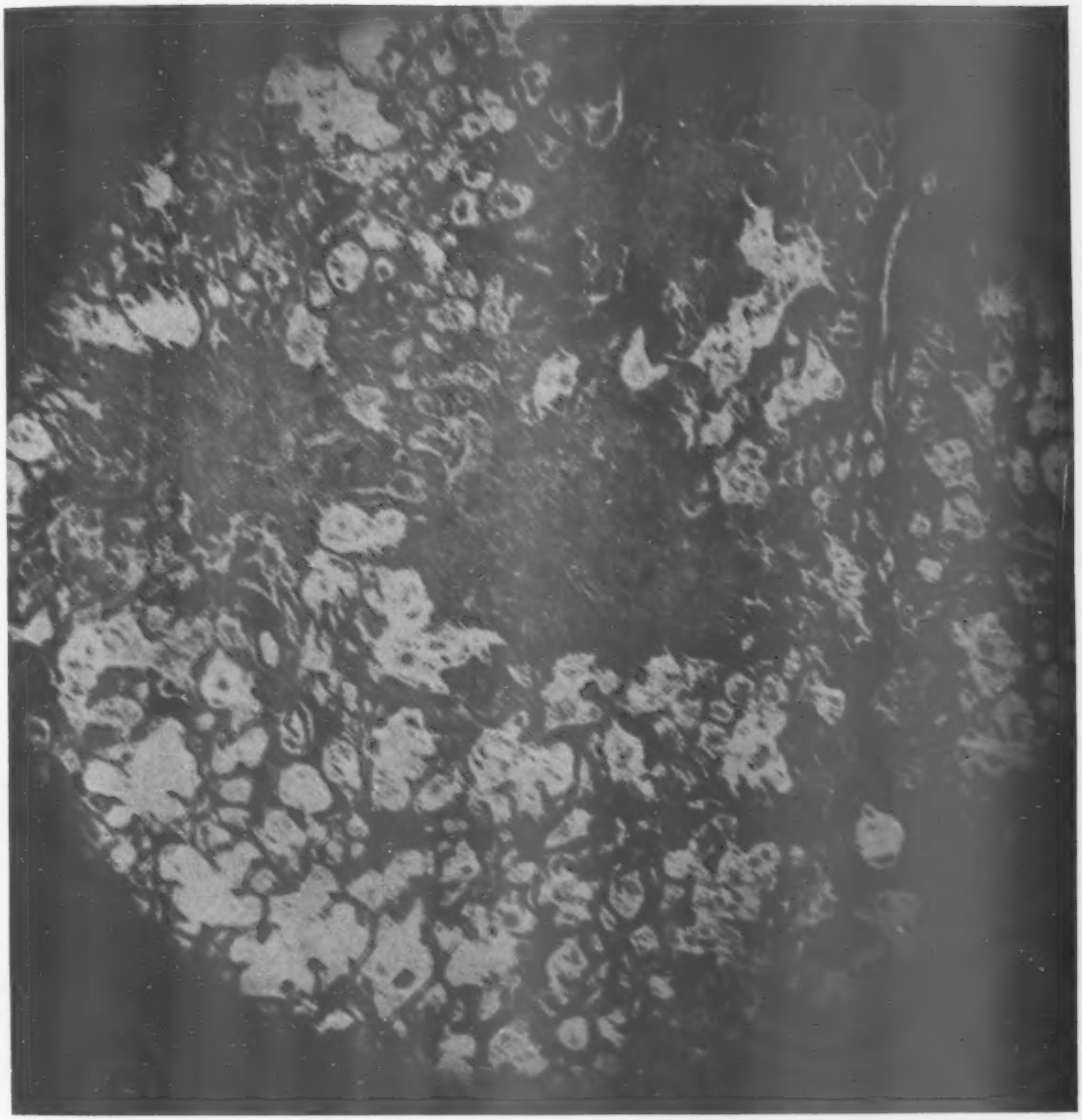

Fig. 51. These small, irregularly spherical masses have the microscopic characteristics of the tubercle of acid-fast bacillus origin, but when they were stained for acid-fast bacilli, none were found but (cont. in Fig. 52)

coniosis, so as to correlate the acute or rapidly developing tuberculoid lesion with the chronic or slowly developing tuberculoid lesion (Fig. 53). The acute dust tubercles (Fig. 51) are relatively free of collagen deposits, whereas the chronic tuberculoid lesion (Fig. 53) has quantities of collagen laid down as whorls surrounded by dust laden phagocytes. These whorls are singularly free of birefractive flecks, as seen by cross polarization (Fig. 54) compared with the surrounding tissue, composed of dust laden phagocytes.

Social and Economic Problems. The social and economic problems of the acute pneumonic parenchymal type and the acute tuberculoid type of pneumoconiosis are the most serious of any of the types of pneumoconiosis because of their rapid development. Indeed, the man may be dead within

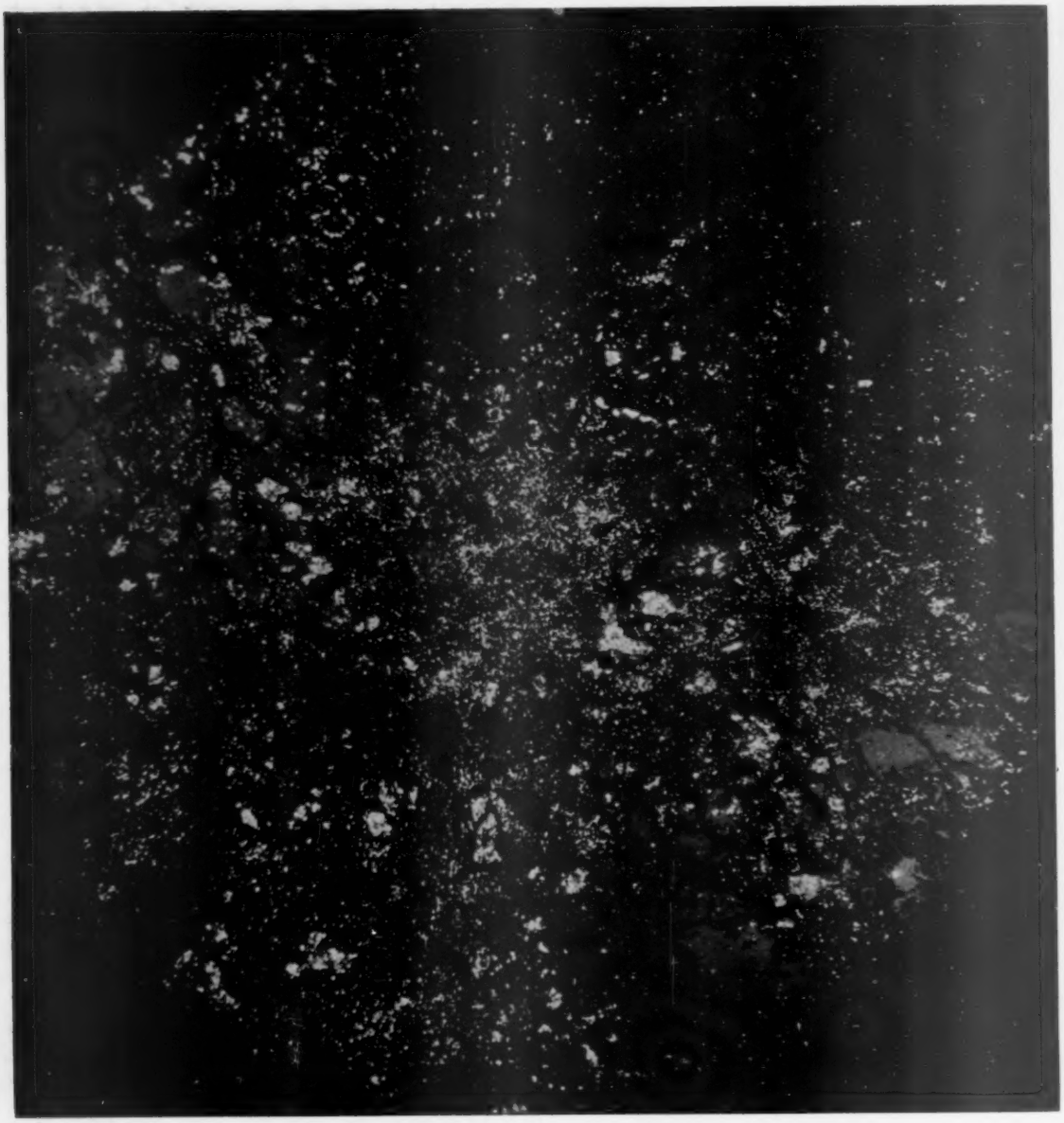

FIG. 52. When the same three tubercles are viewed with a field rendered dark by cross polarization, each tubercle is literally studded with refractive flecks of silica or one of the silicates. This man was dead in less than three years.

two or three years from his first exposure; the patient with the pneumonic parenchymal lesion (Fig. 47) was dead in twenty months; the man with the acute tuberculoid lesion (Fig. 48) was dead in less than three years. These types of lesions are also an important social and economic problem because the roentgen findings are not characteristic of silicosis according to the definition, as they are singularly free of silicotic nodules. For this reason, such lesions may not be recognized as silicosis, and the patient or his family may be deprived of just compensation.

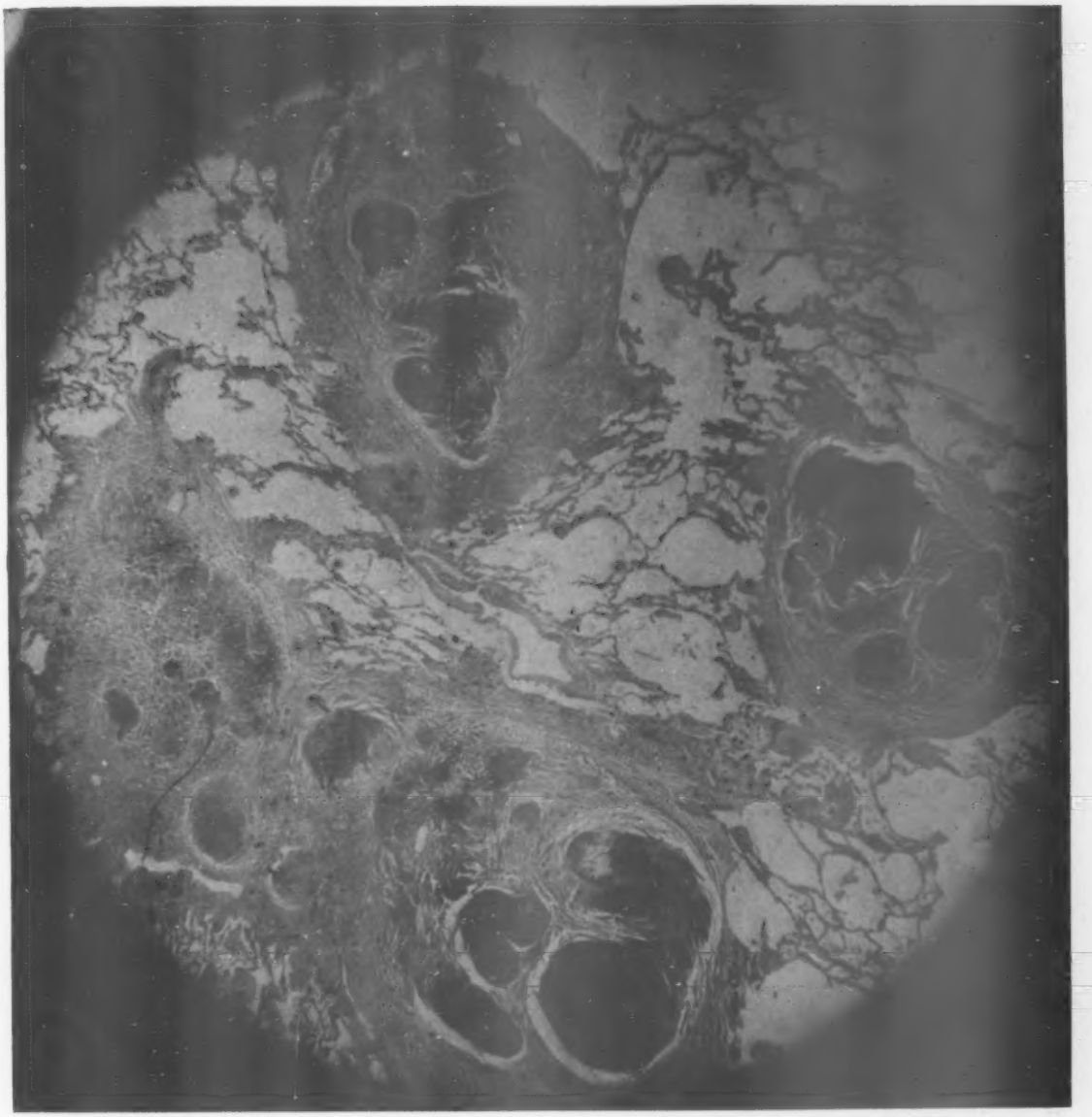

Fig. 53. Likewise, small irregularly spherical masses of long standing have the appearance of old fibrosed tubercles of acid-fast bacillus origin, yet an intensive study by an impartial and unbiased pathologist failed to show any acid-fast bacilli, in a section prepared, cut and stained for this purpose. But when this field was viewed by cross polarization (cont. in Fig. 54)

Vascular changes in pneumoconiosis (silicosis)

Under this heading will be considered the morbid changes that occur within the lumen of the pulmonary arteries and veins, and in their walls, exclusive of the laminated deposits of collagen laid down as a sheath around these blood vessels. In an article entitled, "Dyspnea of silicosis: what causes it?" I expressed my conviction that the dyspnea of which the silicotic patient complains is largely due to vascular changes rather than to deposits of connective tissue in the parenchyma or stroma of the lung,

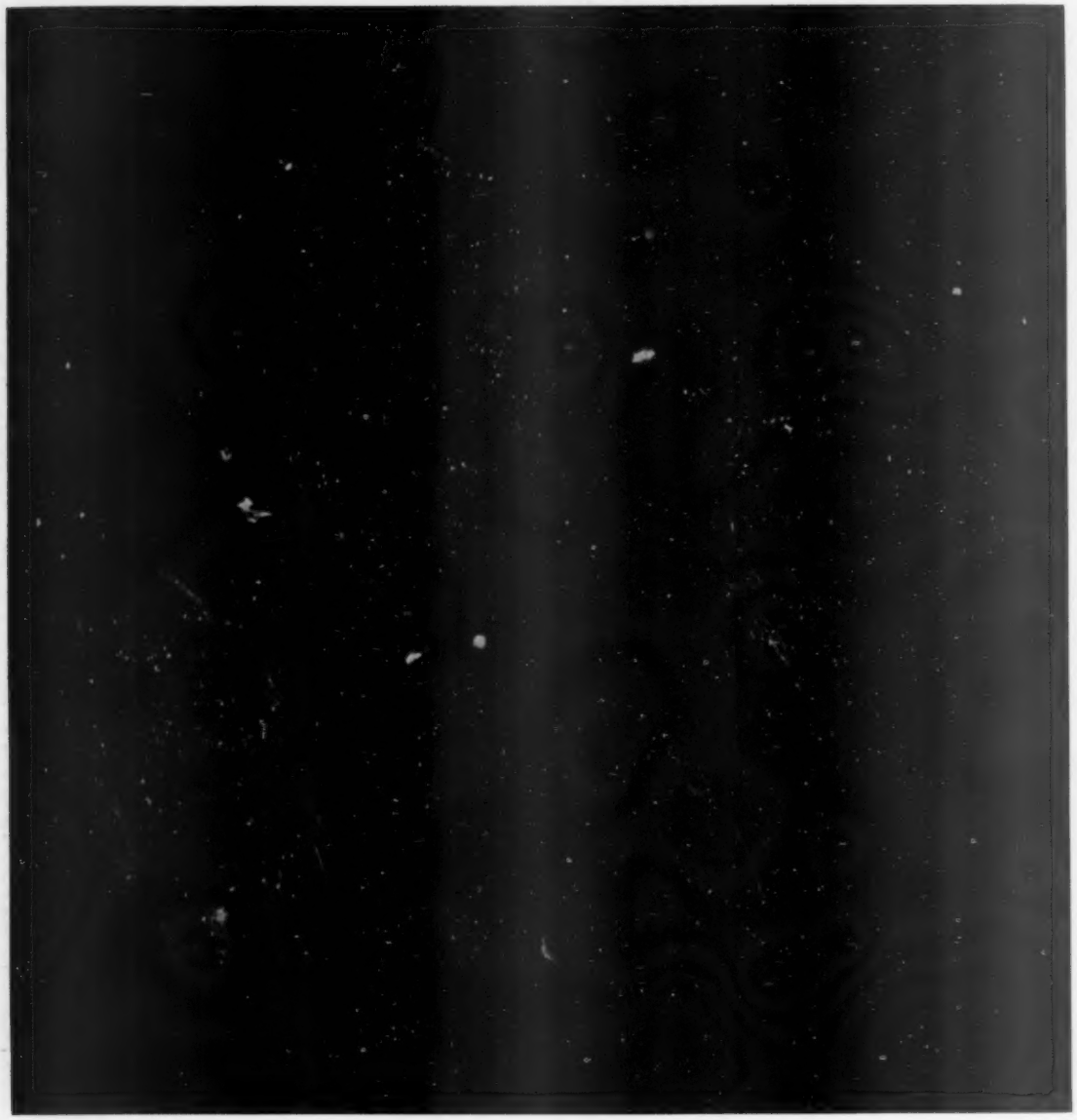

FIG. 54. Quantities of birefractive flecks were discernible. This man had worked for forty years as a granite cutter without a mask and without a blower.

and after four or five years or more intensive study and obtaining very much more data on this subject, I am even more convinced than at the time of presenting the original article. That communication was based upon a study of the acute manifestation of the disease, but subsequent observations have shown even more marked vascular changes to exist in practically all of the microscopic material which I have studied. In the acute cases the vascular changes within the blood vessels were apparently due largely to a slowing down of the circulation of the blood within the avascular areas that was caused by a partial or complete obstruction of the capillary circulation within these avascular areas, with compensating dilatation of the capillaries in the adjacent areas. But the lesions within the lumen of the blood vessel in the

chronic or long standing cases is evidenced by an actual growth of connective tissue within the lumen of the blood vessel, often diminishing its lumen to less than a quarter of its normal diameter (Fig. 55). These connective tissue cells have the aging characteristics typical of connective tissue in that at first they are large, irregularly shaped cells with large nuclei which as they age become fusiform, and still, as they become old, develop into long cylindrical cells with relatively small nuclei. This is one of the most constant findings observed in all of the pathologic material which I have studied.

These vascular changes are very similar to those occurring in coronary occlusion.

Fig. 55. Intravascular growth of reparative connective tissue with cells having aging characteristics have reduced the lumen of the artery to about one-quarter of its normal size. Within the constricted lumen are seen red blood corpuscles and leukocytes. The artery is surrounded by a sheath of collagen, distinctly different from the connective tissue within the artery. An adjacent bronchus surrounded by a sheath of dust laden phagocytes appears in the upper part of the field.

Fig. 56. Roentgenogram of the right lung of a living man with a pulmonary artery injected with diodrast. The roentgenograms were made by Drs. Robb and Steinberg, and were lent to me to show the size, shape and distribution of the pulmonary artery. This roentgenogram was used to make the schematic tracing shown in Figure 20, and it is now compared with (cont. in Fig. 57).

The sudden dyspnea that often occurs late in the course of the disease is similar to coronary attacks. This vascular occlusion or diminution is observed so universally in our pathologic material that it led us to seek to demonstrate the same findings in the living. This thesis was supported by a

Fig. 57. A roentgenogram of a patient with a history of fifty-six years' exposure to dust, complaining of and actually suffering from a moderate degree of dyspnea. Because of the microscopic findings illustrated in Figure 55, an attempt was made to determine whether the diminution in the lumen of the pulmonary arteries could be shown by vasculograms of the pulmonary circulation. Dr. Robb injected the pulmonary circulation in the same manner as in the previous roentgenogram. Various preliminary tests showed a prolonged delay in the diodrast reaching the pulmonary artery, and this finding was amply confirmed by the very marked diminution in the lumen of these arteries, as observed in this roentgenogram, compared with Figure 56, which is normal.

very careful analysis of the clinical history of subjects, where I was able to take the clinical history personally and look for this factor. In one case referred to me by the Mine Workers Union, the clinical history supported this thesis so completely that with the aid of Dr. Robb, I was able to make vasculograms (Fig. 57) of this man showing both the arterial and venous systems of the pulmonary circulation. These vasculograms and the preceding circulatory tests which were necessary to determine the exact instant at which the exposure should be made revealed definite evidence of pulmonary vascular obstruction or stenosis. Roentgenograms were made of both the arterial and the venous systems and the original roentgenograms of the arterial system showed very definite diminution in the diameter of the pulmonary arteries, as compared with similar roentgenograms of the pulmonary artery in the normal subject (Fig. 56) which had been loaned to me by Drs. Robb and Steinberg, whose work on this subject was so epoch making in the study of the pulmonary vascular system. I believe that these vascular changes are less marked in the typical nodular type of pneumoconiosis (silicosis) where the lungs are studded with small, discrete nodules separated by relatively healthy lung than they are in the vesicular type which is relatively free of nodules but where there are deposits of fine strands of collagen involving the terminal blood vessels and bronchioles.

CONCLUSION

As I have said, these highlights and lowlights are extracts from an extensive manuscript prepared for the second edition of the "Story of Dusty Lungs." These findings were observed and recorded, regardless of their significance, or whether they were old or new. No claims are made for original observations, because I have refrained from searching the literature. Some of the findings are conventional and some have been considered unorthodox.

These findings and conclusions presented as they are in this article will give the com-

mentator and critic an opportunity to prove or disprove them, before they are finally published in book form.

The Officers of the John B. Pierce Foundation and the author will welcome any criticism, either constructive or destructive, so that any facts or conclusions herein presented may be retracted or amplified.

56 Greenridge Ave.,
White Plains, New York.

Revelations

Fig. 36. Worked thirty-two years in a mine; positive sputum at the present time.

Fig. 37. History of forty years' exposure to granite dust; no acid-fast bacilli found after three hours' diligent search.

Fig. 38. No exposure to dust; quantities of acid-fast bacilli with growth on culture and positive guinea pig inoculation.

Fig. 39. Tuberculosis of acid-fast bacilli origin.

Fig. 40. Delafield's drawing of pulmonary tubercles of unknown origin.

Fig. 41. Pulmonary tubercles of dust origin; forty years' exposure to granite dust; no acid-fast bacilli found.

Fig. 42. Pulmonary tubercles of acid-fast bacilli origin; quantities of acid-fast bacilli found microscopically.

Fig. 43. History of forty years' exposure to silica dust; no acid-fast bacilli; quantities of silica found microscopically.

Fig. 49. The acute tubercles are of dust origin (transparent, birefractive dust, mostly talc); no acid-fast bacilli found. These pulmonary tubercles are of the same group as shown in Figures 51 and 52.

Fig. 50. The acute tubercles are of acid-fast bacilli origin; no appreciable dust particles present.

DISCUSSION

Dr. William M. Doughty, Cincinnati, Ohio. I had intended to give a more thorough discussion of this paper, but I don't believe anyone could do justice to it without considerable time and, since we have long passed the hour of closing, I shall limit mine to a few remarks. The amount of effort Dr. Cole has unquestionably put upon this subject, not only in covering the slide problem, but in working out the pathological conditions that are actually shown in all of these various types of silicosis, is an advantage that I think all of us interested in this subject will appreciate the more we go into the pathology of it. Dr. Cole, as you all know, is not only a roentgenologist, but a well recognized pathologist, and I feel that his contribution to the pathology of silicosis at this time is really one that most of us interested in the subject will greatly appreciate, and we shall all look forward to the book which he is to publish in the near future.

AERO-OTITIS MEDIA*†

A ROENTGENOLOGIC STUDY

By LIEUTENANT J. C. LARKIN, (MC) USNR

WHEN the air pressure in the tympanic cavity and the auditory tube is not equal to the pressure in the external auditory meatus and the posterior nasal pharynx, there is a progressive sensation of fullness and pain. Armstrong and Heim[1] described this clinical entity and suggested the term aero-otitis media. The results of this inequality of pressure is a collapse of the tympanic membrane against the labyrinthic wall of the internal ear and a shift of the auditory ossicles. This is usually followed by congestion and inflammation of these structures, and there may be hemorrhage into the tympanic membrane from injury to the small vessels. Unless the tensor veli palatini and the salpingopharyngeus are contracted and pulled away from the cartilaginous portion of the tube by yawning or swallowing, the pressure gradient will become so great‡ as the plane descends that the muscles are unable to open the auditory tube. When this happens, or if one has a post-nasal congestion and cannot open the auditory tube, the sudden sensation of fullness is followed by severe pain, vertigo, tinnitus, and at times even nausea. Hearing may be lost for a few hours or even days. If the pressure gradient exceeds one-half an atmosphere the tympanic membrane will usually rupture.

In aero-otitis media the otoscopic examination of the tympanic membrane will reveal it to be retracted, with a partial or complete loss of the light reflex. The vessels running along the handle of the malleus and those in the periphery of the drum will be dilated and hemorrhagic. The handle of the malleus is shorter, since its more distal portion has been carried toward the labyrinthic wall, and one is now looking at it from a more oblique angle (Gleason[4]). In severe cases there will also be a fluid line behind the drum.

As aero-otitis media is rather common among the hundreds of thousands now engaged in aviation, and since individuals so affected are usually grounded temporarily, the disease entity becomes important in aviation medicine. Since the external auditory meatus and the tympanic membrane are the only structures that can be studied and examined by means now available, it would be desirable to find some method to examine the tympanic cavity, the ossicles, the auditory tube, and its pharyngeal opening. It was hoped that roentgen examination of these heretofore not examinable structures might give additional information on the etiology and even possibly the treatment of this condition.

TECHNIQUE OF ROENTGEN EXAMINATION

A modified vertico-submental view is used, similar to the one used in the routine examination for the sphenoidal sinus (Fig. 1). The patient is placed in the position shown in Figure 2 with the chin extended as far as possible. The 23 degree angle board is used to project the structures posterior to the external canal on the bottom of the film. By the use of a 3 mm. aluminum filter to increase the effective wave length, and a 60 cm. cone with a 10 cm. aperture to cut down the scattered rays, the minute structures appear much sharper and detail that would otherwise be blurred is added to the roentgenogram.

ANATOMY

The lateral portion of the external meatus can be visualized on almost any roentgenogram, but the most mesial portion

‡ 8 cm. of mercury.

* From the Department of Radiology, Dispensary, Naval Air Station, Alameda, California.

† The opinions and views set forth in this article are those of the writer and are not to be considered as reflecting those of the Navy Department.

is very indefinite. It is difficult to determine where the external canal ends and the middle ear begins since the tympanic membrane cannot be distinguished on the roentgenogram. It can be determined, however, by injecting lipiodol into the external canal (Fig. 3). The medial end of the external canal forms half an ellipse, with the long axis minus 20 degrees from the perpendicular; this is the line formed by the attachment of the tympanic membrane to the bottom of the external canal. By tracing the outline on a clear piece of film, and by drawing in the other half of the ellipse to indicate its superior and lateral portion the attachment of the entire tympanic membrane is determined. Then by superimposing this ellipse over a film showing the ossicles of the same patient, the exact relation between the ossicles and the drum is determined. Figure 4 is copied from such a drawing and shows the desired relationships. It is found that the ossicles of the ear lie above and lateral to the central portion of the tympanic membrane, and only the handle of the malleus and the long process of the incus actually extend over the central portion of the drum. The tympanic

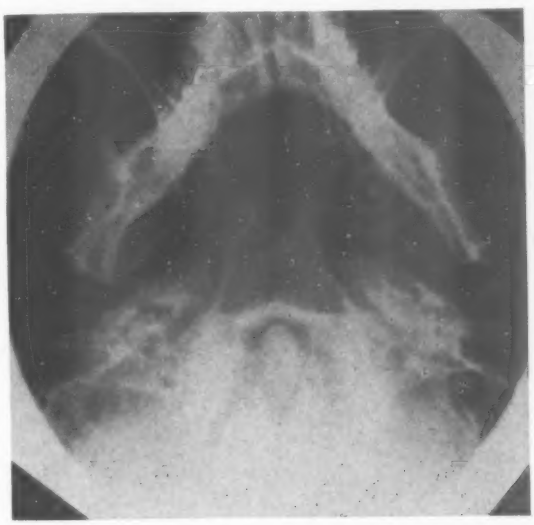

Fig. 1. The routine position for demonstration of the sphenoid sinus. The ossicles, auditory tube, and the pharnygeal ostium can be seen on the left side.

Fig. 2. Position for taking the vertico-submental view. The central longitudinal axis of the cone passes in a line just anterior to the external auditory meatus and posterior to the angle of the jaw.

Fig. 3. Lipiodol in the canal to outline the medial aspect of the external auditory meatus. Note the cochlea, *C*, with its three turns. The canal for the facial nerve, *F*, is also seen. This demarcates the region of the semicircular canals, *S*.

membrane cuts under the point where the stapes attaches itself to the fenestra vestibuli of the inner ear. This is shown by the broken line of Figure 4. Fortunately this gives air background for the stapes to be visualized on the roentgenogram. In the routine position (Fig. 1) the long axis of the ossicles are perpendicular to the film, so

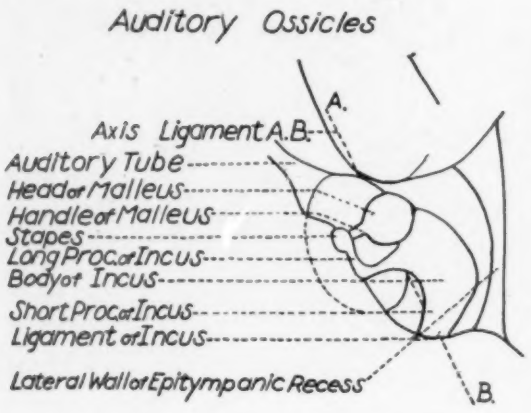

FIG. 4. Drawing to show the relation between the ossicles and the tympanic membrane and also the partition between the middle ear and the external canal.

there is the greatest possible density of bone to absorb the roentgen rays. The head of the malleus and a portion of its neck are almost completely on end so that they cast the densest shadow (Fig. 5). Projecting out from the head, running mesially and posteriorly at an angle of 45 degrees, is the handle of the malleus. The body of the incus is much less dense than the head of the malleus, but casts a shadow several times larger. It is attached to the head of the malleus and has a short and a long process. The short process, the most lateral portion of the ossicle structures, is attached through the ligament of the incus to the wall of the rigid epitympanic recess to form the posterior portion of the axis ligament (Cunningham[3]). The anterior portion of the axis ligament is formed by the anterior ligament of the malleus. The rigid ossicles on rotating about this axis, *AB*, carries the impulses from the tympanic membrane to the stapes. The stapes can be seen on some roentgenograms as a minute projection over the tympanic membrane from the labyrinthic wall of the inner ear. It is the most mesial structure seen, and is situated above the handle of the malleus. On the roentgenogram, however, they appear to be connected. The structure seen just lateral to the ossicles is the lateral wall of

FIG. 5. The auditory ossicles of the ear enlarged about six times. Drawing of Figure 4 is made from this roentgenogram.

the epitympanic recess, and it seems to actually surround the outer half of the ossicles. The dark area mesial to the ossicles is the tympanic cavity and the beginning of the auditory tube.

The bony portion of the auditory tube beginning with the middle ear has a rigid wall, measures about 1.5 cm. long, and can always be seen on the roentgenogram. The cartilaginous portion of the tube is 2.5 cm. long. Its superior and mesial surfaces are rigid, while surfaces on the inferior and lateral sides are made up of the levator veli palatini and the tensor veli palatini respectively. The auditory tube then runs in

Fig. 6. The course of the auditory tube. Note the funnel-shaped opening of the auditory tube in the posterior oral pharynx.

a direction of about 45 degrees to the posterior nasopharynx, in front of the foramen lacerum and behind the foramen spinosum

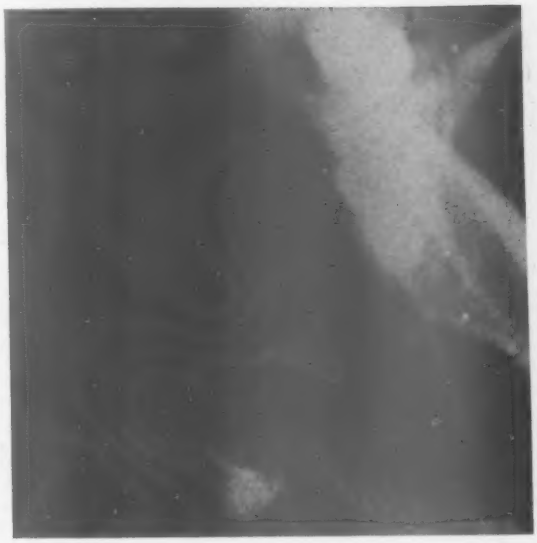

Fig. 7. Lipiodol in the pharyngeal ostium of the auditory tube. The posterior oral pharynx and the membranes have shrunk down on this side.

and ovale. This cartilaginous part of the tube runs parallel to the groove between the greater wing of the sphenoid and the petrous portion of the temporal bones. In some cases the separation is so wide a sphenoid-petrous fissure is formed. But in any case there may be a narrow line on the

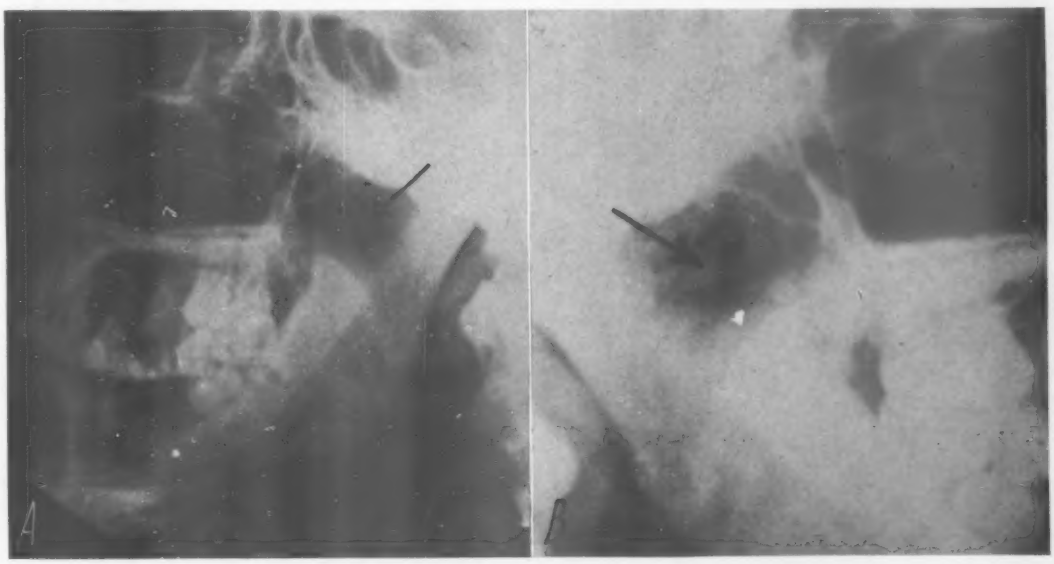

Fig. 8. Pharyngeal ostium of the auditory tube as seen in the lateral view. The opening can be seen best when the right and the left ostia are superimposed.

roentgenogram which may easily be mistaken for the auditory tube. However, the tube is usually wider and more lateral, and there can be no mistake in its identity when it is seen to end in the funnel-shaped pharyngeal ostium (Fig. 6). The junction between the cartilaginous and bony portions unfortunately is obscured by the spine of the sphenoid bone. The part that enters the posterior oral pharynx runs beneath the medial pterygoid plate, and therefore it also cannot always be seen. Between these two obscure points, indicated by arrow in Figure 11, Case 1, is a shadow measuring 2 mm. wide and 20 mm. long which is the cartilaginous portion of the auditory tube distinguishable on the roentgenogram and called the "Tubenknorpel" (Corning[2]).

The exact position of the pharyngeal ostium of the auditory tube and its shape and contour are determined by placing lipiodol in the ostium (Fig. 7). The ostium is also seen in the routine lateral view taken to show the soft tissues of the neck (Fig. 8). It is necessary to cocainize the posterior nasopharynx to insert the cannula, and in so doing there is shrinking of the mucous membranes. The ostium is actually a vertical slit, but when seen from above (Fig. 7) it appears to be funnel shaped. The lateral border of the ostium in Figure 7 is the superior portion as seen in Figure 8, and the mesial border is the inferior portion seen in the lateral view. The auditory ostium is located on each side of the posterior nasopharynx, just anterior to the fossa of Rosenmüller and posterior to the inferior nasal meatus.

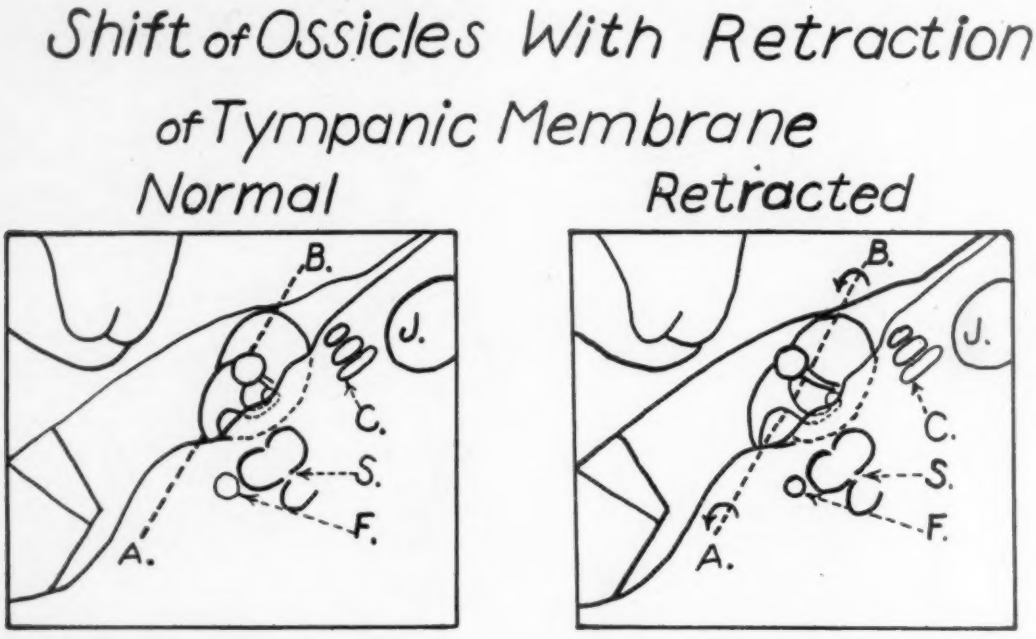

FIG. 9. Drawing of the auditory ossicles when the tympanic membrane is in normal position. *C*, the cochlea; *F*, the facial canal; *S*, the semicircular canals. These structures are seen so seldom on roentgenograms that neither their presence nor their absence has any diagnostic significance.

FIG. 10. The position of the ossicles after retraction of the tympanic membrane. The ossicles have rotated about the axis, *A B*, and have assumed a new position more anterior and more lateral than is seen in Figure 9.

POSITION OF THE TYMPANIC MEMBRANE

The auditory ossicles are opaque, and since they move with the tympanic membrane, it is possible to determine the position of the membrane indirectly by the

position assumed by the ossicles. Figure 9 shows the axis, *AB*, and the position assumed by the ossicles in relation to the other structures. When the handle of the malleus is carried inward by the retraction of the tympanic membrane, the rigid ossicles must also move; but since they rotate about the axis *AB*, the head of the malleus and the parts of the ossicles above the axis must move in a direction just opposite to that of the handle. It then assumes a position seen in Figures 10 and 15, Case v. It is noted the distance between the head of the malleus and the stapes is increased, and as a result the handle of the malleus appears longer in Figure 10 than in Figure 9. Also, when this happens, since the axis *AB*, is at an oblique angle to the auditory canal, the ossicles must also move across the tympanic cavity to a new position that is both more anterior and also more lateral, as is shown in the drawings.

PRESENCE OF FLUID IN THE MIDDLE EAR

This condition, known as hydrops ex vacuo, is thought to result from the creation of partial vacuum in the ear, causing a change in the osmotic relations and resulting in a flow of lymph into the middle ear. This can at times be seen as a fluid line upon examination of the drum with the otoscope. The ossicles are, however, very often found to be cloudy on the roentgenograms, since only a small amount of exudate is needed to obscure their outlines. Thus small amounts of fluid can be detected which would be difficult to find by other methods.

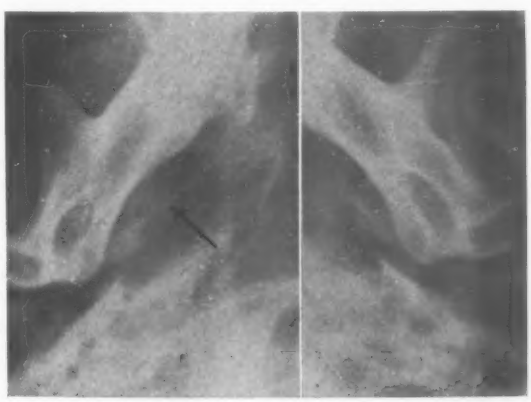

Fig. 11. On the left side the ossicles and surrounding air cells are cloudy, indicating aero-otitis media. The head of the malleus has assumed a more lateral and anterior position. The cartilaginous part of the tube is quite prominent on the injured side.

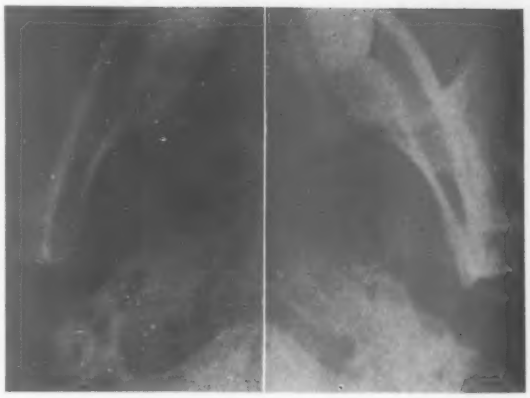

Fig. 12. The hazy ossicles and surrounding air cells on the right side together with the shift of the ossicles indicates an aero-otitis media. On the left side there is also a slight shift, but there is no indication of fluid and congestion.

CLINICAL APPLICATIONS

Methods have now been worked out for the examination of the middle ear, the position of the tympanic membrane and its relation to the ossicles of the ear, the determination of fluid in the middle ear, and the patency of the auditory tube. These methods of examination are now applied to some of the conditions found on examination of flyers suffering from injuries to their auditory mechanisms. It is also possible to study the effect of drugs on these structures.

CASE HISTORIES

Case I (Fig. 11). This dive-bomber pilot received his injury two weeks ago. The right side has completely recovered but he still has a feeling of fullness in the left ear.

Case II (Fig. 12). A dive-bomber pilot, aged twenty-two, who on a dive-bomber mission developed bilateral blockage on his first dive from 14,000 feet. He was able, however, to get air into the left auditory tube, but the right remained blocked. On examination the right

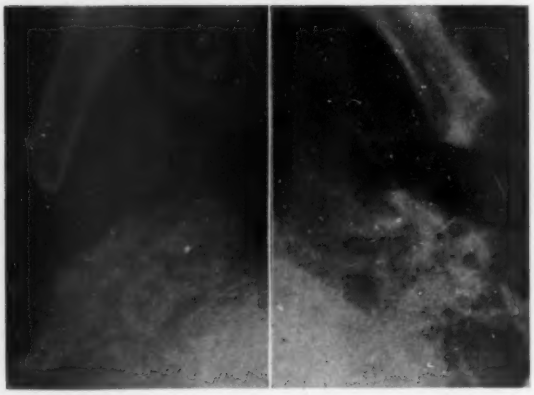

FIG. 13. The head of the malleus has shifted almost to the lateral wall of the epitympanic recess, and there is a small amount of clouding on the right side. The stapes is unusually prominent. Left side is normal.

drum was scarlet, and the left appeared to be normal.

CASE III (Fig. 13). A transport pilot who on descending developed bilateral blockage. On examination the left drum is normal. The right shows the typical appearance of aero-otitis media.

CASE IV (Fig. 14). A dive-bomber pilot developed bilateral aero-otitis media after completing his mission two hours before. Most of the dives were made from a height of 15,000 feet. He has had chronic aero-otitis media for the past two years.

CASE V (Fig. 15). An A.M.M. 3/c developed pain in the right ear while in the low pressure chamber on being returned to normal atmospheric pressure. The air had been reduced to a pressure corresponding to that found at a height of 35,000 feet.* He was able to insufflate his left auditory tube, but the right remained blocked.

CASE VI (Fig. 16). An S. 2/c developed aero-

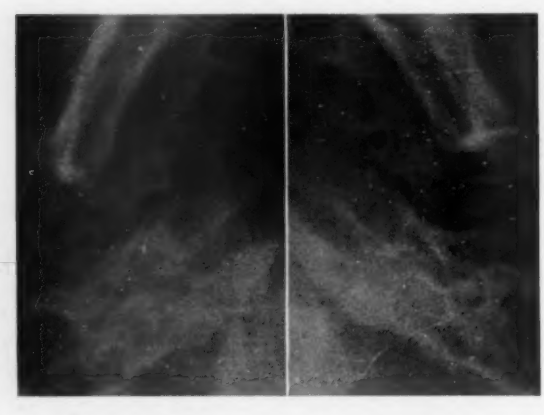

FIG. 14. The ossicles are obliterated on both sides. The cartilaginous part of the tube is well visualized on the left side.

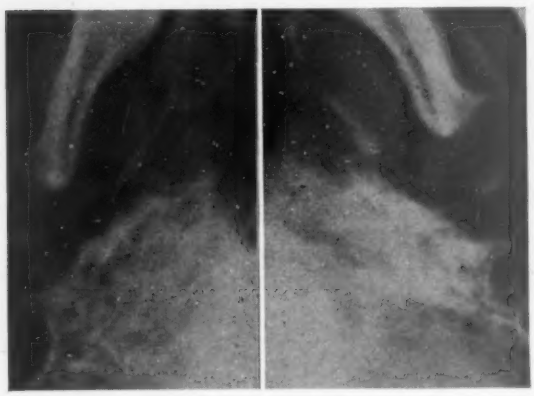

FIG. 15. The right side shows a shift of the ossicles similar to that in drawing of Figure 10, while on the normal left side the ossicles are similar to those of drawing, Figure 9. There is also slight clouding on the injured right side.

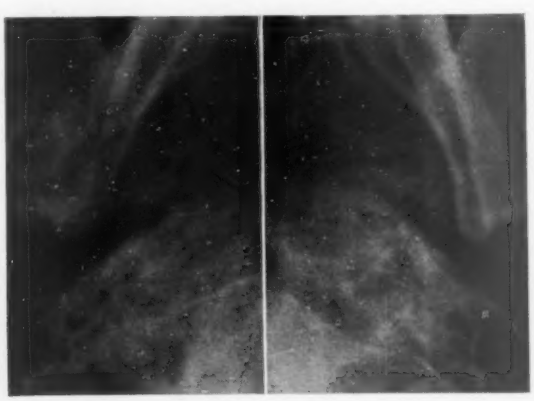

FIG. 16. The ossicles are cloudy and shifted laterally on the left side, and the auditory tube is prominent. The right side is normal.

otitis media after an indoctrination run in the low pressure chamber. All his symptoms are on the right side, and the left side is asymptomatic.

* The incidence of aero-otitis media is quite high in men being indoctrinated in flying activities in low pressure chambers.

DISCUSSION

Unfortunately the auditory structures are not always demonstrable on the roentgenograms, so that their presence or absence is of no clinical value; and it is only when the right and left sides can be compared that the examination yields useful information. The finding of abnormally placed ossicles in cases having no symptoms suggests that position is not an absolute criterion for symptomatic aero-otitis media. However, the blurring of the ossicles due to congestion and the accumulation of exudate in the tympanic cavity and the surrounding air cells show correlation with the clinical findings. This roentgen study reveals for the first time the presence of fluid in the cells of the petrous bone and in the cells of the mastoid region in cases of aero-otitis media. This is due to hydrops ex vacuo and is a result of fluid oozing from the mucous membrane lining the air cells and covering the auditory ossicles. The presence of this fluid in the air cells probably accounts for the sensation of fullness experienced.

When the tensor palatini contracts it changes the normally collapsed auditory tube into one with a wide lumen. Since it is common to find the shadow of the "Tubenknorpel" on one side only (Case 1, Fig. 11), and since the two sides are identical anatomically, the unilateral shadow on the roentgenogram must be due to air in the lumen. Thus this part of the auditory tube could not prevent the passage of air into the tympanic cavity. Three cases of aero-otitis media reported here do have such tubes, on the affected side, and hence the constriction must be at the ostium.

SUMMARY

Methods have been worked out for the roentgen examination of the external auditory meatus, the shifting of the tympanic membrane as shown by the position assumed by the auditory ossicles, the presence of fluid in and about the tympanic cavity, and the patency of the auditory tube.

The above methods have been applied to a number of cases of aero-otitis media in an attempt to study the mechanism of the injury and to find a possible remedy.

It is found that the position of the auditory ossicles is not an absolute criterion for the diagnosis of aero-otitis media. However, the clouding of the auditory ossicles and of the surrounding cells in the petrous bone and mastoid areas due to the presence of fluid indicates aero-otitis media.

In aero-otitis media the auditory tube is probably blocked, usually in the pharyngeal ostium.

The use of 10 per cent cocaine causes a marked shrinking of the mucous membranes of the posterior oral pharynx and of the pharyngeal ostium. This can be demonstrated on the roentgenogram.

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THE EFFECT OF ROENTGEN RAYS ON THE JOINT EFFUSIONS IN CERTAIN NONSPECIFIC ARTICULAR LESIONS IN HUMANS, AND ON THE NORMAL JOINTS OF DOGS

A PRELIMINARY REPORT

By THOMAS HORWITZ, M.D.,* *and* MELVIN A. DILLMAN, M.D.†
PHILADELPHIA, PENNSYLVANIA

IT IS NOT claimed, as a result of these clinical and experimental observations, that roentgen therapy is or is not beneficial in the treatment of certain arthritides. It is felt, however, that, in the doses administered, this form of therapy has been valuable *in the control of the joint effusions* found in certain nonspecific synovial lesions involving the knee joints in humans, and that, within the recommended doses, there have been no immediate or as yet remote deleterious effects. The longest observation period in this group has been from October 12, 1940, to the present (February, 1942). The more vulnerable articulations of the extremities of dogs, treated with much larger doses than have been utilized and found effectual in human subjects, have shown no immediate and as yet no remote ill effects on the articular and para-articular structures. The longest observation period in the animals has been from December 16, 1940 to the present (February, 1942). No attempt has been made to duplicate, in animals, the nonspecific synovial lesions encountered in the human subjects, because of the absence of known specific criteria as to the etiology and nature of these articular lesions.

USE OF ROENTGEN THERAPY IN THE CONTROL OF THE JOINT EFFUSIONS IN CERTAIN NONSPECIFIC SYNOVIAL LESIONS INVOLVING THE KNEE JOINTS IN HUMANS

One knee joint in 3 subjects and both knee joints in 3 others were treated—a total of 9 knee joints. In these 6 cases no other joints than the knees were involved. In 2 patients the effusions were intermittent. In 1 subject (F.C.) the phenomenon occurred in one knee, and, after eleven months of recovery in this knee following roentgen therapy, appeared in the opposite knee, and in the other patient (S.G.) the intermittent effusion occurred in only one knee. In the interval periods between attacks, the involved knees in these 2 cases were anatomically and functionally normal, and asymptomatic. In 3 patients the synovial effusion was constantly present—in 1 subject (W.M.) occurring in one knee and appearing, five months after recovery of that knee following roentgen therapy, in the opposite knee, and in the other patients (M.D. and J.B) the knee involvement being unilateral. In the youngest patient (F.S.), there were continuous effusions present simultaneously in both knees. In the 4 cases with constant knee effusions, aspirations of the affected joints restored, temporarily, a full range of painless motion, but revealed some residual thickening of the joint lining in 3 subjects. The only complaint in our 6 cases was that of knee effusion, with incidental "sense of fullness" in the joint and impaired range of motion. Extensive clinical laboratory examinations—including urinalyses; blood count, blood sedimentation rate, blood Wassermann and Kahn tests and complete blood chemistry estimations; cytologic, bacteriologic, serologic and streptococcus agglutination studies of the joint fluid; intracutaneous tuberculin test and

* Corinna Borden Keen Research Fellow in the Department of Anatomy, Jefferson Medical College, Philadelphia.
† Roentgenologist assigned to the Department of Anatomy, Jefferson Medical College, Philadelphia.

roentgenographic studies—were uniformly normal or negative.

The etiology could not be definitely ascertained, the onset being gradual and without apparent cause in all of the subjects, with the possible exception of the young patient (F.S.) who had suffered an attack of rheumatic fever prior to the onset of her knee involvement. The diagnosis in the 2 cases with recurrent knee effusions was "intermittent hydrarthrosis" and in the 4 cases with continuous effusions was "chronic nonspecific synovitis with effusion." In all cases, previous treatment by repeated aspirations, compression bandages, external fixation and intensive physiotherapy, and, in 2 cases, nonspecific protein therapy, had failed to alter the clinical picture.

The methods of treatment and the results in these cases are given in Table 1.

Synovial biopsy, under local anesthesia, was performed through a 2 inch linear anteromedial incision, prior to or during roentgen therapy in both knees of 2 cases (F.C. and W.M.), and in one knee prior to treatment in a third case (M.D.), in an effort to determine the nature of the synovial lesions and to ascertain the modus operandi of roentgen therapy in the control of the joint effusions. The slight variations in the technique of deep roentgen therapy were due to the necessity of our employing two different machines during the investigation.

Results of Roentgen Therapy in the Human Subjects.

Clinical. There were no untoward reactions evident under the fractionated therapy, even in those receiving two full courses of roentgen therapy to the knee joint, except in 1 case (J.B.) where a slight erythema was observed after the first course had almost been completed (1,296 roentgens to the inner and 1,296 roentgens to the outer aspects of the knee). The effusion in this case failed to respond to this series, and the patient did not return for further treatment. The 8 knee joints treated in the other 5 patients showed favorable responses during the first or second course of roentgen therapy. The full effect was observed in 7 of the 8 knee joints at or just prior to the termination of the full course. In the right knee of W.M., complete recovery was not observed until one month after the completion of the course of roentgen therapy, suggesting that in this instance some benefit may have accrued from cumulative roentgen-ray effect. In the right knee of F.C. only a slight response followed one course of roentgen treatment, but complete recovery followed a biopsy procedure without further roentgen therapy. Although this might suggest a possible beneficial effect of the arthrotomy *per se*, the evidence is otherwise in the other instances where a biopsy was performed. Its use *prior* to roentgen therapy was without apparent clinical benefit in the left knee of the same patient, in the right knee of W.M., and in another case with chronic effusion of one knee, not herein reported, who failed to return for roentgen therapy.

In F.C., there has been complete recovery from recurrent effusions in both knees that are, at present, asymptomatic and anatomically and functionally normal. The same result has been obtained in W.M., whose knees were the site of chronic joint effusions. In one knee in M.D. and in both knees in F.S., the constant synovial effusions have disappeared and the joints are asymptomatic, although there is some residual synovial thickening in each instance. In S.G., although the distressing recurrent effusions no longer occur in the left knee, there is some residual synovial thickening and discomfort on weight bearing.

Roentgenographic. There has been no evidence, thus far, on repeated roentgenographic examinations, of change in the osseous and para-osseous structures of the treated and control knee joints. The longest period of observation has been from October 12, 1940 to the present (February, 1942).

TABLE I

ROENTGEN THERAPY FOR CERTAIN NONSPECIFIC SYNOVIAL LESIONS OF THE KNEE JOINT IN HUMANS

Case	Sex	Age	Knee Joint Involved	Nature of Effusion	Duration	Date Treatment Begun	Time of Biopsy in Relation to Therapy	Factors in Roentgen Therapy (two fields: inner and outer surfaces of knee)	Total Dose (roentgens)	Results to Date
F.C.	F	54	Left	Intermittent. Every 10 days and lasting 4 days	27 yr.	Oct. 16, 1940	Before	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 11 min.; each dose 198 r; 7 weekly treatments to both fields	1,386 to each field	No effusions and complete anatomical and functional recovery, since Nov., 1940 or 3 wk. prior to termination of roentgen therapy
			Right	Intermittent. Every 10 days and lasting 4 days	3 wk.	Sept. 23, 1941	After	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; 6 weekly treatments to both fields	1,290 to each field	Only partial diminution in severity of intermittent effusions following roentgen therapy; complete recovery following biopsy, Dec., 1941
S.G.	F	22	Left	Intermittent. Every 2 wk. and lasting 6 days	5 yr.	June 17, 1941	None	Course 1: kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 11 min.; each dose 198 r; 4 weekly treatments to inner and 3 weekly treatments to outer fields. Followed by: kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; 3 weekly treatments to inner and 4 weekly treatments to outer fields. Treated biweekly, but only once weekly to each field, for 7 weeks	Course 1: 1,437 to inner and 1,455 to outer fields	Complete recovery from intermittent effusions. Residual pain and slight synovial thickening (since Nov., 1941)
								Course 2: kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r. No. of treatments as in Course 1. Interval between two courses, 10 weeks	Course 2: 1,505 to each field	
W.M.	M	51	Right	Continuous	1 yr.	Oct. 12, 1940	Before Course 1	Course 1: kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 11 min.; each dose 198 r; treated biweekly, once weekly to each field, for 7 weeks	Course 1: 1,386 to each field	No effusion and complete anatomical and functional recovery, since April, 1941 or one month after termination of Course 2
								Course 2: as Course 1. Interval between two courses, 6 weeks	Course 2: 1,386 to each field	
			Left	Continuous	2 wk.	Aug. 12, 1941	Following Course 1 and preceding Course 2	Course 1: kv. 190; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; treated biweekly, once weekly to each field, for 7 weeks	Course 1: 1 505 to each field	No effusion and complete anatomical and functional recovery since termination of Course 2 in Jan., 1942
								Course 2: as Course 1. Interval between two courses, 6 weeks	Course 2: 1,505 to each field	
M.D.	F	58	Right	Continuous	1 yr.	June 17, 1941	Before Course 1	Course 1: as Course 1 in Case S.G.	Course 1: 1,437 to inner and 1,455 to outer fields	Disappearance of all effusion at termination of Course 2 in Nov., 1941. Slight residual synovial thickening
								Course 2: as Course 1 in Case S.G.	Course 2: 1,505 to each field	
J.B.	M	31	Right	Continuous	9 mo.	Nov. 18, 1941	None	Kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; biweekly treatments, once weekly to each field, for 6 weeks	1,296 to each field	No diminution of effusion. Single course not completed due to slight erythema. Did not return for second course
F.S.	F	13	Bilateral and Simultaneous	Continuous	4 yr.	Oct. 21, 1941	None	Course 1: kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; three biweekly treatments, once weekly to each field of both knees treated simultaneously	Each knee—Course 1: 645 to each field	All effusion disappeared in both knees after termination of Course 2 in Dec., 1941. Residual asymptomatic synovial thickening in both knees
								Course 2: as Course 1. Interval between two courses, 6 weeks	Course 2: 645 to each field	

It is anticipated, although not as yet evident, that early epiphyseal growth arrest may be initiated in the knee regions of the thirteen year old subject, even with the minimal therapeutic doses used. This will not be considered untoward in this case if it occurs, since the dosage has been administered equally to both knees, and because the child has already equaled the height of her parents.

Histopathologic. Synovial biopsy was performed *prior* to any treatment in 3 cases (M.D., F.C.—left knee; W.M.—right knee). The pathologic features bore no constant relation to the clinical features in these 3 cases and for this reason an attempt to classify the clinicopathologic features in our cases has not appeared worth while. For example, the clinical features of M.D. and W.M. were similar in that there was asymptomatic, continuous swelling of the knee, with normal laboratory and roentgenographic findings, and in each subject recovery followed biopsy and roentgen therapy, except for some residual synovial thickening in M.D. The histopathologic features, however, were not alike. In both biopsy specimens the synovial layer of cells appeared normal, and the pathologic changes were confined to the subsynovial tissues that were edematous and very vascular. In M.D. there was a very diffuse sprinkling of monocytes, lymphocytes, plasma cells and red blood cells throughout the subsynovial connective tissue, while in W.M. there were numerous focal perivascular collections of chronic inflammatory cells. This latter histopathologic picture was present also in F.C., even though in this case the swelling was not constantly present but was regularly intermittent. In F.C. the laboratory and roentgenographic findings were also normal, and the recovery following biopsy and roentgen therapy was complete. Special fat stains in these 3 specimens showed only scattered lipoid deposits.

In 2 cases, synovial biopsy was performed *after* one full course of roentgen therapy (1,290 r to the inner and 1,290 r to the outer sides of the right knee of F.C., and 1,505 r to the inner and 1,505 r to the outer aspects of the left knee of W.M.). In both cases there had already been evidence of improvement. The biopsies, however, were performed to secure histopathologic material for comparison with the specimens removed from the opposite knees of these cases prior to roentgen therapy. The specimens removed *following* roentgen therapy showed the surface synovial cells to be normal. The subsynovial tissues still demonstrated numerous perivascular collections of chronic inflammatory cells, but illustrated *three additional features:* (1) disappearance of edema, (2) a diffuse fibrosis and (3) thickening of the vessel walls with obliteration of the lumina of the smaller vascular channels. There were no areas of tissue degeneration or tissue death. In W.M. there were many large macrophages, filled with phagocytosed blood pigment granules, in the perivascular lymphocytic collections (Fig. 1 and 2).

ROENTGEN THERAPY TO THE NORMAL PERIPHERAL JOINTS OF DOGS

Six dogs, all male hounds about one year old and in apparently perfect health, were utilized in this study. The areas surrounding the treated joints were protected during roentgen therapy and the comparable joints of the opposite limbs were used as untreated controls. The left knee (stifle) and elbow joints were treated in all 6 animals, while in dog No. 3 the right hip was also treated and the left hip used as the untreated control. These dogs were kept in cages and the functional stress on their limbs was kept to a minimum.

An effort was made to study the effects of the following factors which have been evaluated in Table II:

1. The *type* of roentgen therapy—superficial or deep (always fractionated).

2. The *dose* of each roentgen treatment, and the total amount of roentgen-ray dosage.

3. The *duration* of active treatment, the duration of the cumulative period, and the

TABLE II

ROENTGEN THERAPY TO THE NORMAL JOINTS OF DOGS

Dog No.	Weight (lb.)	Date Treatment Begun	Joint Treated	Type of Therapy	Factors of Therapy (single field: medial aspect of joint)	Total Dose (roentgens)	Duration Treatment (weeks)	Duration Cumulative Period (weeks)	Effects		
									Clinical	Roentgenographic	Histopathologic
4	25	Dec. 23, 1940	L. knee (stifle joint)	Superficial	Kv. 135; ma. 5; skin target distance 30 cm.; filter 4 mm. glass; time 4 min.; each dose 200 r, every 2nd day for 15 treatments	3,000	4	8	None	None	None
			L. elbow	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, each 2nd day for 15 treatments	3,000	4	2	None	None	None
1	12	Jan. 11, 1941	L. knee	Superficial	Kv. 135; ma. 5; skin target distance 50 cm.; filter 4 mm. glass; time 10 min.; each dose 500 r, every 2nd day for 12 treatments	6,000	4	4	Loss of hair and superficial ulceration	None	None
			L. elbow	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 20 min.; each dose 400 r, every 2nd day for 15 treatments	6,000	4	2	None	None	None
5	20	March 2, 1941	L. knee	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, every 2nd day for 20 treatments	4,000	6	12	None	None	None
			L. elbow	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, every 2nd day for 10 treatments	2,000	3	8	None	None	None
3	14	Feb. 24, 1941	L. knee	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, every 2nd day for 20 treatments during Course 1 and daily for 20 treatments during Course 2	8,000 Course 1—4,000 Course 2—4,000	11 Course 1—6 Course 2—5	8 Between courses—5 Between Course 2 and death—3	None	None	None
			L. elbow	Deep	Above factors; each dose 200 r; two courses of 10 treatments each, administered every 2nd day	4,000 Course 1—2,000 Course 2—2,000	6 Course 1—3 Course 2—3	11 Between courses—8 and death—3	None	None	None
			R. hip	Deep	Above factors; each dose 200 r; one course of 15 treatments, administered every 2nd day	3,000 (only one course)	5	4	None	None	None
2	16	Aug. 4, 1941	L. knee	Deep	Kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 200 r, daily for 20 treatments	4,000	3	From Aug. 28, 1941 to Feb., 1942	None	None	Still living
			L. elbow	Deep	Above factors; each dose 200 r, daily for 10 treatments	2,000	10 days	From Aug. 15, 1941 to Feb., 1942	None	None	
6	14	July 17, 1941	L. knee	Deep	Kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 400 r, daily for 10 treatments	4,000	10 days	From Sept. 29, 1941 to Feb., 1942	None	None	Still living
			L. elbow	Deep	Above factors; each dose 400 r, daily for 5 treatments	2,000	5 days	From Sept. 29, 1941 to Feb., 1942	None	None	

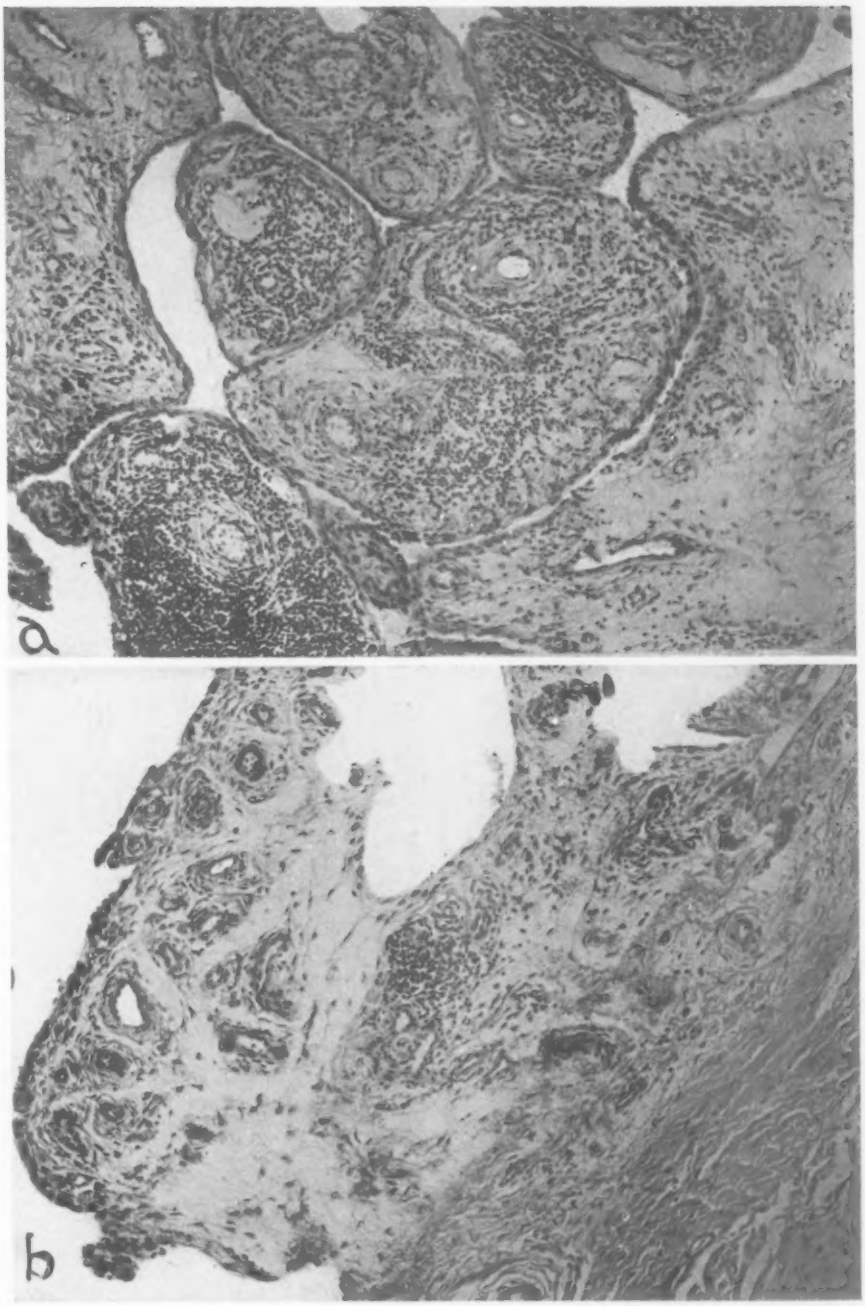

Fig. 1. *a*. Photomicrograph (×110) of synovia of right knee of patient F.C., *prior* to roentgen therapy. The surface cells lining the closely packed synovial villi are normal, but the subsynovial tissues demonstrate considerable edema, many dilated blood vessels and many focal perivascular collections of chronic inflammatory cells.

b. Photomicrograph (×110) of synovia of left knee of the same patient, *following* a course of roentgen therapy (1,290 r to inner and 1,290 r to outer fields). The surface cells show no considerable change. There is less edema of the subsynovial tissues, the vessels are no longer dilated and the lumina of several vessels have become obliterated, while there is only one collection of chronic inflammatory cells in the entire field.

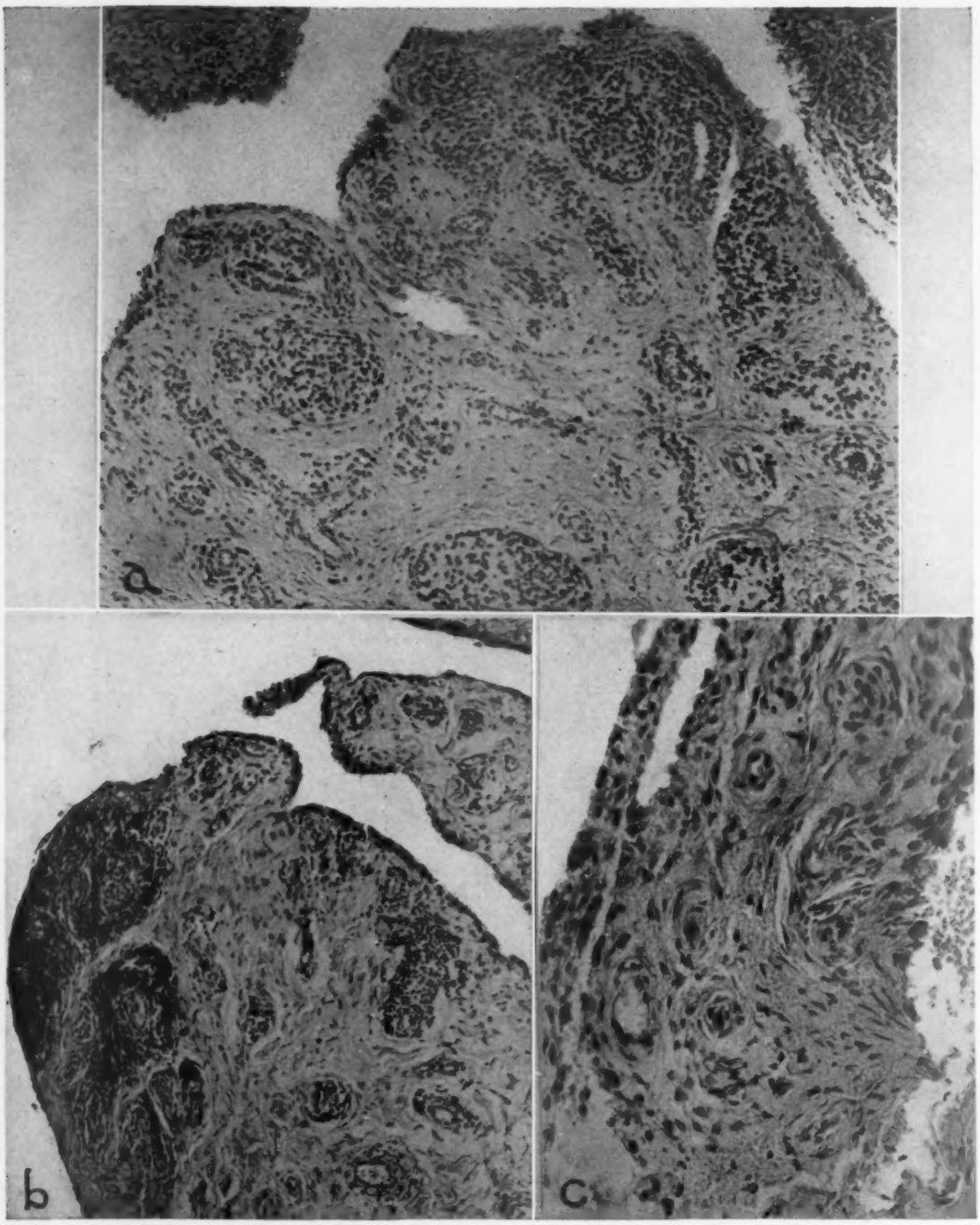

FIG. 2. *a*. Photomicrograph (×110) of synovia of right knee of patient W.M., *prior* to roentgen therapy. The surface lining cells appear normal. The subsynovial tissues are edematous, very vascular, and contain a diffuse sprinkling and many focal perivascular collections of chronic inflammatory cells.

b. Photomicrograph (×110) of synovia of left knee of the same patient, *following* a course of roentgen therapy (1,505 r to inner and 1,505 r to outer fields). The surface cells stain deeply but show no abnormal changes. The subsynovial tissues show a diffuse fibrosis, the edema has disappeared, the vessels are no longer dilated and are less numerous, and, in some instances, show obliteration of their lumina. There are scattered, small perivascular collections of lymphocytes, and to the left of the section, these contain many macrophages that are filled with blood pigment granules.

c. Photomicrograph (×220) of another area of the synovia of the irradiated left knee of W.M. This illustrates the marked fibroplasia in the subsynovial tissues, the thickening of the vessels walls, and the narrowing and obliteration of the lumina of some of the vascular channels.

number of treatment-series and interval (cumulative) periods.

Roentgen therapy was administered only to the inner aspect of the joint, because this side was more easily approached and because the small dimensions of the peripheral articulations in the dogs did not appear to require bilateral fields. Dogs No. 4, No. 1, No. 5 and No. 3 were killed after variable periods following the completion of roentgen therapy. The treated and control articulations were roentgenographed prior to and following the full course of therapy, and, at death, these joints were removed for histopathologic study. Dog No. 3 received the longest course of treatment (three and one-half months), the largest total dose to any one joint (8,000 r to the left knee joint), the largest combined total dose to all of the treated joints (8,000 r to the left knee joint, 4,000 r to the left elbow joint and 3,000 r to the right hip joint), and the longest cumulative periods. In order to evaluate "delayed roentgen-ray effect" and the effect of time more critically, dogs No. 2 and No. 6 are being observed for three and five years, respectively, following their courses of roentgen therapy.

Results of Roentgen Therapy in the Dogs.

Clinical. Only dog No. 1 showed loss of fur and some superficial skin ulceration over the left knee that had been subjected to superficial therapy. None of the dogs, including No. 1, evidenced any apparent discomfort in the treated joints up to their death. They manifested no limp and no apparent pain or tenderness; there was no swelling, and the passive and active ranges of motion remained unimpaired. This has also been true in the 2 living dogs which have been observed from the onset of their treatment on July 17 and August 4, 1941, to the present (February, 1942).

Roentgenographic. There were no changes in the roentgenograms of the treated and control joints made prior to treatment and just before death. Nor have there been any changes, thus far, in roentgenograms of the joints of the living dogs.

Pathologic. Gross examinations and histopathologic studies of the treated and control joints in dogs No. 4, No. 1, No. 5 and No. 3, including the articular cartilage, subchondral bone, synovial tissue, joint capsule and adjacent muscle tissue, revealed no abnormal changes in any of the specimens and no differences between the treated and control joints (Fig. 3, 4, 5, 6 and 7).

DISCUSSION

Roentgen therapy was utilized, with success, to control the effusions in a case of intermittent hydrarthrosis of the knee by May, Huet and Barnaud[20] (1937), and by Epstein and Edeiken[9] (1940). No recurrence was observed by the former authors in their case for ten months, nor by the latter authors in their case for twenty-six months. Intermittent hydrarthrosis is characterized by a periodically recurring effusion in the affected joint, of several months' duration, with intervals in which the joint is symptom free and functionally normal. The association of intermittent hydrarthrosis with rheumatoid arthritis has been observed by several authors (Krida[14]). In the experience of Ghormley and Cameron,[12] instances of "chronic synovitis" and of "intermittent hydrarthrosis" are in reality manifestations of chronic infectious arthritis, for in their cases other joints became affected subsequent to synovectomy. Cecil[7] has seen several cases of chronic intermittent hydrops develop typical rheumatoid arthritis. Inge[21] has also seen cases of chronic monarticular villous synovitis prove to be the precursors of generalized rheumatoid arthritis. For this reason no effort has been made to classify the joint involvement in our cases.

The use of roentgen rays in the treatment of the arthritides is not being evaluated in this study. Although recent, carefully controlled, clinical studies indicate that this form of treatment is of no value in rheumatoid arthritis involving the joints of the extremities (Smyth, Freyberg and Peck[24]), but is of value in the relief of symptoms in rheumatoid arthritis of the

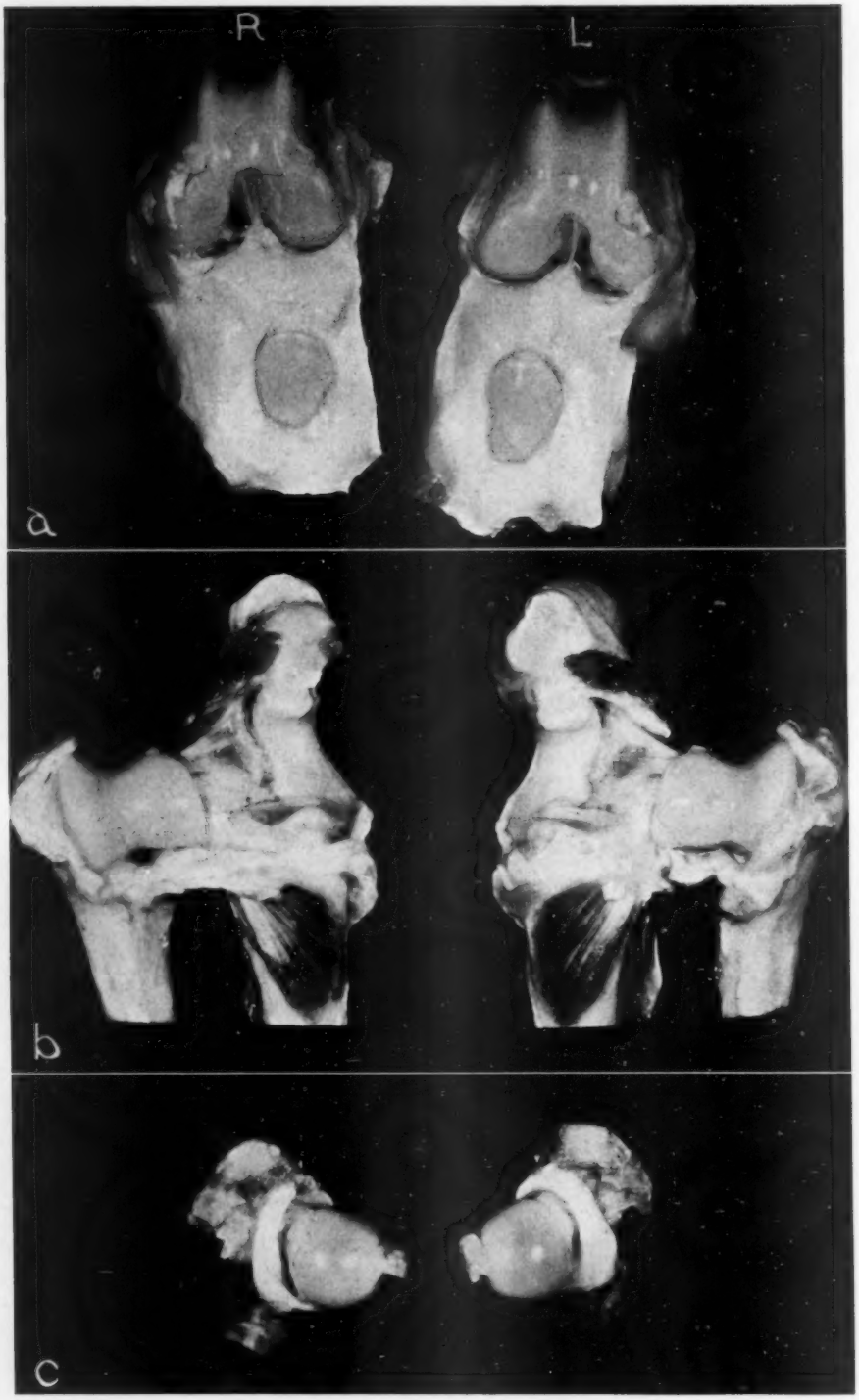

FIG. 3. Dog. No. 3. *a*, left knee joint (*L*) treated with 8,000 r (deep therapy) and untreated right knee joint (*R*). *b*, left elbow joint (*L*) treated with 4,000 r (deep therapy) and untreated right elbow joint (*R*). *c*, right hip joint (*R*) treated with 3,000 (deep therapy) and untreated left hip joint (*L*). There are no gross differences in the osseous and para-osseous tissues of the treated and control joints.

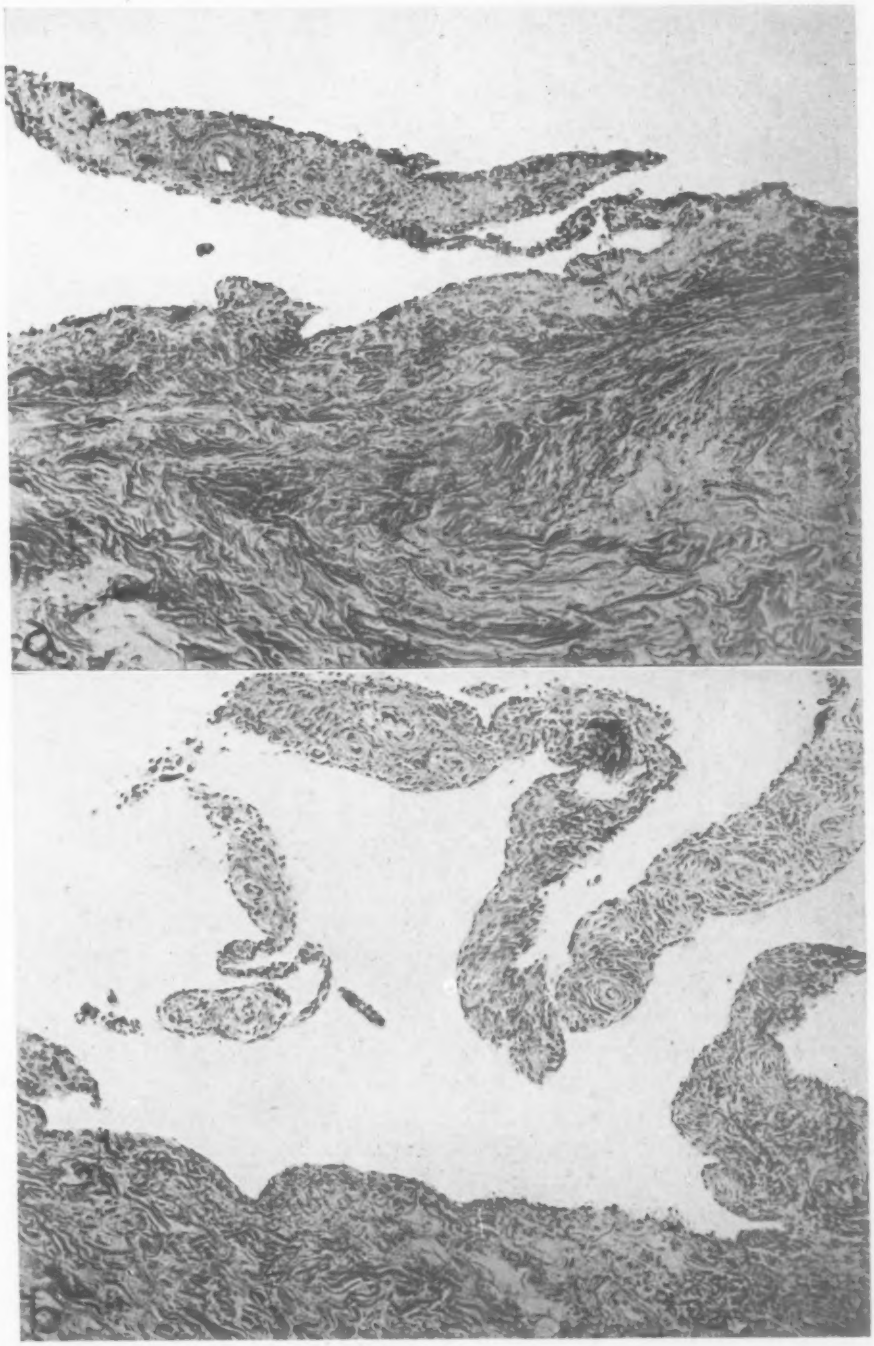

Fig. 4. Dog. No. 3. *a*, photomicrograph (×110) of the synovia of treated left knee joint (Fig. 3*a*). *b*, photomicrograph (×110) of the synovia of control (untreated) right knee joint (Fig. 3*a*). The histologic findings are normal and identical in both specimens.

spine (Smyth, Freyberg and Lampe[23]), no adequate explanation has been offered for the modus operandi in these cases.

Although the joint effusion and its attendant discomfort have been eliminated in the 8 affected knee joints in 5 of our 6 cases, there is no reason to suggest that the roentgen therapy in these cases will also

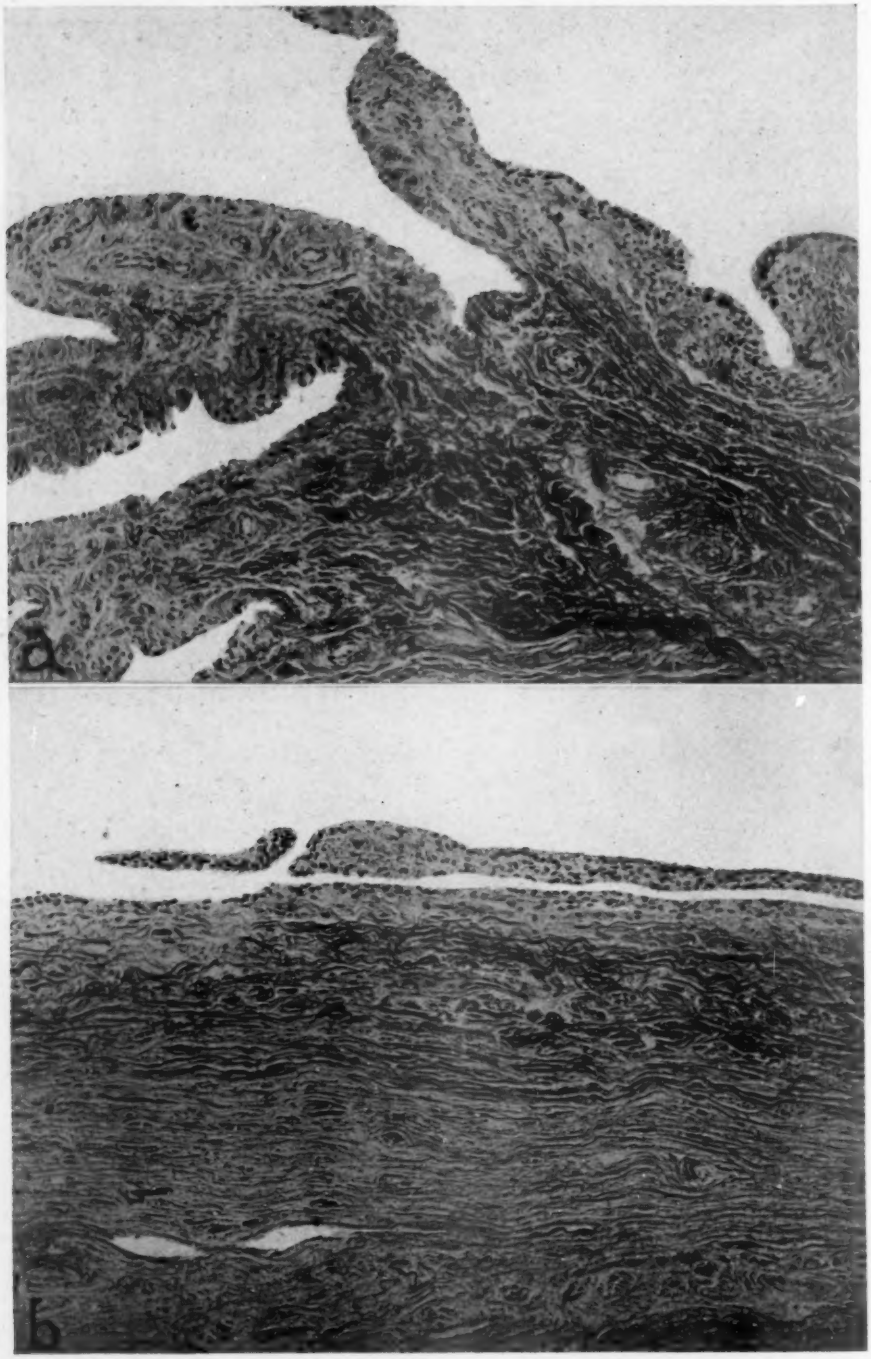

FIG. 5. Dog. No. 3. *a*, photomicrograph (×110) of the synovia of treated left elbow joint (Fig. 3*b*). *b*, photomicrograph (×110) of the synovia of control (untreated) right elbow joint (Fig. 3*b*). The histologic findings are normal and identical in both specimens.

serve to favorably affect the progress and ultimate course of the underlying disease processes. Indeed, the residual asymptomatic synovial thickening in the treated joints of M.D. and F.S., and the painful synovial thickening in the irradiated knee

Fig. 6. Dog. No. 5. *a*, left knee joint (*L*) treated with 4,000 r (deep therapy) and untreated right knee joint (*R*). *b*, left elbow joint (*L*) treated with 2,000 r (deep therapy) and untreated right elbow joint (*R*). There are no gross differences in the osseous and para-osseous tissues of the treated and control joints.

Fig. 7. Dog. No. 4. *a*, left knee joint (*L*) treated with 3,000 r (superficial therapy) and untreated right knee joint (*R*). *b*, left elbow joint (*L*) treated with 3,000 r (deep therapy) and untreated right elbow joint (*R*). There are no gross differences in the osseous and para-osseous tissues of the treated and control joints.

joint of S.G., indicate the persistence of the pathologic joint lesions in these cases. We believe that certain features in the pathologic specimens removed from 2 cases following roentgen therapy may represent some of the responsible factors in the con-

trol of the joint effusions by this form of treatment. These features are: (1) the disappearance of edema, (2) the increase in fibrosis and (3) the thickening of the vessel walls and obliteration of the smaller vascular channels in the subsynovial tissues.

The great sensitivity of vascular endothelium is one of the most important factors in radiation therapy. The mode of action of radiation on tumors, for example, is generally believed to be due partly to the direct action on a certain proportion of the tumor cells, and partly to the damaging effect on the circulation (Pohle,[21] Warren[27]). While lymphoid cells, polymorphonuclear leukocytes, and epithelial cells are very sensitive, connective tissue, muscle, cartilage and bone tolerate large exposures to radiation (Desjardins[8]), except such as may be interpreted as interfering with their blood supply. Changes in the lymph flow probably play a similar rôle but are difficult to demonstrate (Ewing[10]). In bone, such vascular changes may cause late bone necrosis, appearing months after treatment has ceased (Ewing,[11] Watson and Scarborough[28]). The neck of the femur has been found to be particularly vulnerable to the vascular changes incidental to roentgen therapy for pelvic malignancy (Strauss and McGoldrick[26] and others[1,3,13]).

The experiments of Lacassagne and Vinzent[19] and Lacassagne[16,17,18] demonstrated the occurrence of rabbit sarcoma in regions of acute inflammation, induced by injecting *Streptobacillus caviae* and exposed to irradiation by roentgen rays. There have been a number of clinical reports of sarcoma following roentgen therapy of human joints, the pathologic lesion prior to treatment being definitely tuberculous in some of the cases (Küttner,[15] Becker[4]). Because the malignant changes did not become manifest in these reported cases for several years, our clinical material, as well as the living dogs No. 2 and 6, will be observed for three to five years following treatment.

The total dosage in our human cases has been kept low, and has been fractionated. Despite the experience that much larger doses applied to the more vulnerable joints of our dogs were without immediate and, thus far, remote deleterious effects, it is our experience that very heavy roentgen-ray dosage will affect normal human joints adversely. Painful and disabling swelling, loss of motion and roentgenographic evidence of a destructive arthritis have been observed in the normal knee joint adjacent to a heavily irradiated benign osseous lesion involving the medial femoral condyle in a twenty-nine year old female, and the upper tibia in a fifteen year old boy. In a third case, a seventeen year old male with a benign osseous lesion in the lower end of the femur that had been heavily irradiated, the adjacent, partially ankylosed knee joint was examined histopathologically following its excision for the correction of a flexion deformity.* It was observed that the synovia had suffered little or no change. There was increased vascularity and a diffuse sprinkling of chronic inflammatory cells in the subsynovial tissues, with a tendency to the formation of focal perivascular collections. The articular cartilage had suffered considerable change—in some areas being thinned or absent, in others being frayed, or calcified, or covered by granulation tissue (pannus). The subchondral bone was very porotic, with increased marrow fibrosis. Despite all of these degenerative manifestations there was no evidence of malignant transformation. The benign tumor tissue in this specimen was well removed from the articular tissues and it was interpreted that these articular changes had followed the intensive roentgen therapy to the juxta-articular tissues.

Bisgard and Hunt[5] and others[6,22] have observed retardation of bone growth in young laboratory animals following irradiation of the epiphyseal plates. Barr, Lingley and Gall[2] observed that within a dosage of 1,000 to 1,335 r (200 kv.) sufficient damage occurred to the epiphyseal plate to cause

* Histologic specimens in this case were examined in the Laboratories of Pathology of the Hospital for Joint Diseases, New York City; Director, Dr. Henry L. Jaffe.

growth retardation of about 50 per cent in laboratory animals, but that within this range the effects noted in the articular cartilage, synovia, skin and muscle were negligible, and the bone marrow changes appreciable but localized. These experiments have been extended as a therapeutic measure by Spangler[25] to secure epiphyseal growth arrest in 3 children and he has concluded that around 3,000 r, given in one fractionated series, will produce epiphyseal closure in humans. This author observed no clinical evidence of soft tissue change or damage, and no roentgenographic evidence of alteration in bone trabeculation in the irradiated areas as late as nine months after treatment. These observations lend support to our present belief that roentgen therapy, in the doses administered by us, is without deleterious effect to human knee joints that are the site of nonspecific synovial lesions and to the normal joints of the extremities of dogs.

CONCLUSIONS

Roentgen therapy has been found beneficial in the *control of the joint effusion* and its attendant discomfort in certain nonspecific synovial lesions involving the knee joint in humans. Within the recommended doses, no immediate and, as yet, no remote deleterious effects have been observed in these cases. The effusion was eliminated in eight knee joints in five patients, and no change was effected on the synovial effusion of the knee joint in the sixth case following an incomplete course of roentgen therapy. Residual asymptomatic synovial thickening has persisted in three knee joints of two patients and painful synovial thickening in the knee joint of one patient. There is, therefore, nothing to suggest that roentgen therapy has served or will serve to affect favorably the progress and ultimate course of the underlying pathologic processes in these cases.

The more vulnerable articulations of the extremities of six normal adult dogs, treated with much larger doses than have been utilized and found effectual in the human subjects, have shown no immediate, and, thus far, no remote untoward effects.

The effect of such factors as type, dose and duration of roentgen treatment have been analyzed in this preliminary report. "Delayed roentgen-ray effect" and the time factor must be evaluated in the human subjects and in the two living dogs, and these findings will be presented in a subsequent report.

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STEREOSCOPIC DEPTH PERCEPTION*

By OSCAR V. BATSON, M.D., *and* VIRGINIA E. CARPENTIER, R.N.
PHILADELPHIA, PENNSYLVANIA

WHILE TIME and thought have been spent on the theory of stereoscopic roentgenography and upon the development of technical apparatus, unfortunately little attention has been given to the personal ocular equipment of the radiologist. The state of the radiologist's own ocular and oculomotor apparatus, and the degree of his ability to fuse stereoscopic images into a satisfactory three-dimensional whole are commonly unknown quantities. Arguments over interpretation of stereoroentgenograms might disappear if all persons viewing them had the same development of stereoscopic vision. One of us (Batson) has been interested for a long time in the way in which physicians and medical students respond to the roentgen stereoscope. Some staunchly maintain that the three-dimensional image can be made to appear as if viewed from the posterior or the anterior aspect at will. This ability to interchange the appearance of the image disappeared in three persons after their vision was corrected. With such experiences in mind it was thought advisable to investigate the exact state of the stereoscopic vision of our trainees in roentgenology in the Graduate School of Medicine. The cooperation and approval of the Vice Dean for Roentgenology, Dr. George E. Pfahler, and of the then Vice Dean for Ophthalmology, the late Dr. William T. Shoemaker, were readily obtained. The first group of student-physicians was examined in the academic year 1941–1942, in the Orthoptic Clinic of the Graduate Hospital, under the direction of the junior author. Examinations were carried out again this year (1943) on the group at present enrolled.

It is obvious that a physician with little or no depth perception can obtain slight benefit from stereoscopic roentgenograms. Such a one should be cognizant of his own deficiency, and might possibly limit his examinations to "flat" roentgenograms. Further, a defective stereoscopic sense might be improved by the use of orthoptic exercises or correction of any existing refractive error.

Three groups of tests were used. First, a check was made of the visual acuity and muscle balance. The visual acuity of the physician was determined both with and without his usual optic correction, if any. One of these examined was wearing a wholly inadequate correction. He realized that he should consult his ophthalmologist, but he had no idea that he was depending largely upon one eye.

The group was next tested for the ability to fuse the right and left eye images into a single whole. All of those tested had this ability; in fact, all had what is technically known as "gross third degree fusion." That is to say, all could see a pair of stereoscopic pictures of a box or a barrel as a three-dimensional object. This test is carried out with suitable cards, and a Brewster type stereoscope. Since all of our group had two functioning eyes, it might be assumed that long before entering medical school fusion of this degree would have been developed. Roentgenology is probably not a specialty favored by anyone with the handicap of only one functioning eye.

It should be emphasized that all of those examined were familiar with the roentgen stereoscope, and had had some experience in its use. All thought that they were able to use the stereoscope satisfactorily, because they could see a chest "in the round." This ability would be expected since all had "gross third degree fusion." Some knew that they could not see all of the details in stereoroentgenograms that would be de-

* From the Department of Anatomy, Graduate School of Medicine, and the Orthoptic Clinic, Graduate Hospital, University of Pennsylvania, Philadelphia, Pennsylvania.

scribed by others, but did not believe that this was due to any deficiency in their personal visual equipment.

The third group of tests was the most significant since it determined the amount of critical depth perception, known technically as stereopsis. This is the faculty necessary to perceive depth relationships—important in accurate roentgen interpretation. The degree of stereopsis was determined with suitable image cards, and a hand stereoscope. In these tests the Kestone stereometric cards were used, and the lower the percentage, the poorer the stereopsis. Seventy per cent of stereopsis is considered the low limit of normal, while provision is made for ratings as high as 110 per cent. Not knowing the minimum percentage required in roentgenologic interpretation, we decided to accept 70 per cent as normal.

Binocular vision is the ability to use both eyes simultaneously to produce a single mental impression. In "third degree fusion" this faculty is developed to the extent that the slightly dissimilar and separate images, as perceived by the two eyes, can be blended into one which has a sense of depth or perspective. Fusion is a cerebral function and is first manifest about six to eight months after birth. This depth sense is developed through use. The more this fusional ability is used, the more perfect will fusion become. It is not clear how late in life one may increase the fusional ability, hence we note the ages of those examined.

Imperfect balance of the extraocular muscles, that is a tendency of the visual axis of each eye to deviate from that of the other, will naturally impair fusion and stereoscopic perception. Also, errors of refraction contribute to difficulty in fusion in several ways. If one eye is much more nearsighted or farsighted than the other, or if one eye is nearsighted and the other farsighted, the images on the two retinae will be of different sizes and degrees of sharpness. These images will therefore be difficult to fuse. Uncorrected astigmatic errors cause distortion, and hence dissimilarity of the two retinal images. The farsighted have blurred images at the near point. The nearsighted individual may find that since he does not have to use his accommodation fully at the near point, the convergence-accommodation ratio is so disturbed that fusion becomes difficult. When the age is reached that the observer depends upon bifocal glasses to supplement his accommodation, he must be trained to use his convergence to the same degree as formerly. Proficiency in depth perception decreases if this is not done.

Another cause for a low percentage of stereopsis in an otherwise normal individual is lack of attention. This individual will have to learn to use this power of observation.

In all, 16 physicians were examined. Ten had average, or better than average, depth perception. The ages of the 10 ranged from twenty-six to thirty-nine years; the percentage of stereopsis from 73 to 107 per cent. There was no age correlation. At the time of examination, no uncorrected refractive errors were found in this normal group. None needed exercises for muscle imbalance.

The other 6 physicians presented enough individually interesting features to warrant a brief summary of each case.

Case I, aged twenty-nine, had one degree of stereopsis. By correction of his muscle balance, with the practice use of prisms, this doctor was able to bring his stereopsis up to 30 per cent. His problem was discussed with him, and he was advised to continue the investigation further, when impending military duty permitted. Even 30 per cent stereopsis should be of an advantage.

Case VIII, aged twenty-seven, had a stereopsis of 35 per cent. After a two hour period of explanation, and experimentation with fusion cards, he brought his perception up to 88 per cent. In other words, he had normal ocular and oculomotor equipment, but had never learned to use it in the manner required in the test. He may have no further difficulty now that he is conscious of the problem.

Case X, aged thirty-two, is one of the most interesting. When examined in January, he

had a stereopsis of 45 per cent. He began the daily use of exercise cards, but it was not until July that he brought his stereopsis up to 90 per cent. He believed that his ability to interpret stereoscopic roentgenograms increased concurrently. Unfortunately, the significance of the test percentages in terms of roentgen interpretation is not known. It may be necessary for this physician to continue the use of the exercise cards to maintain his improvement.

CASE XII, aged forty, when first examined did not have a proper correction of his refractive error. His stereopsis was 25 per cent. After refraction his stereopsis still remained at 25 per cent. Following his second examination, he used the exercise cards proper for his type of muscle imbalance; when again retested ten days later his depth perception was 100 per cent

CASE XIII, aged thirty, had 10 per cent stereopsis, but following a thirty minute period of explanation and experimentation, this became 90 per cent.

CASE XIV, aged thirty-nine, stereopsis 28 per cent, was, like the preceding case, able to bring this to 97 per cent after a one-half hour instruction period.

To summarize, 10 out of a group of 16 beginners in roentgenology had adequate depth perception. Three more with normal oculomotor and ocular apparatus had not come to appreciate the significance of seeing stereoscopically, at least not to the degree required in the test used. A short practice period increased their test percentage to normal range. The remaining 3 did not improve with a short practice period. One of these required ten days, one six months. The remaining case, on whom we were unable to complete our studies, had a defect in muscle balance which could be partially corrected by the practice use of prism exercises. This man expects to follow through at a later date.

It is a pleasure to acknowledge the interest and the close cooperation of the entire group undergoing these tests. Although the correlation between test and roentgen practice is not known, from the diagnostic and remedial results obtained with this small group, we venture to suggest that all beginners in radiology have the exact state of their stereoscopic vision determined. The daily use of the roentgen stereoscope is in itself good stereoscopic training, yet established roentgenologists might well avail themselves of an examination of their stereoscopic efficiency. This examination would be especially revealing at the age period when near vision begins to recede.

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THE ROENTGEN DEMONSTRATION OF MECKEL'S DIVERTICULUM*

By M. H. POPPEL, M.D., F.A.C.R.
NEW YORK, NEW YORK

ALTHOUGH numerous and adequate descriptions of Meckel's diverticulum have appeared in the literature, the illustrations accompanying these papers usually fail to demonstrate the pouch very clearly.

This case is presented because it represents a classical visualization of a Meckel's diverticulum. Fortunately, the entire diverticulum, its neck and the ileum proximal and distal to the diverticulum, are completely visualized, enabling one to gauge the size of the pouch, its shape, position, relation to the small bowel, and its distance from the ileocecal valve.

The omphalomesenteric or vitelline duct (the duct of communication between the yolk sac and the primitive digestive tube in early fetal life) normally disappears within a few weeks after birth, but it occasionally persists more or less as a fibrous cord, a tube or a diverticulum. In the latter instance, the diverticulum is due to the persistent patency of the proximal or mesenteric end of the duct. Anatomically, it is found in about 1 to 3 per cent of individuals and is usually situated 1½ to 3 feet above the ileocecal region in the cadaver. It springs from the antimesenteric border, and its blind end is usually free or it may be connected to the abdominal wall or some other portion of the bowel. Usually, the diverticulum has the same morphology as the ileum, but occasionally aberrant tissue, such as gastric mucosa or pancreatic tissue, has been found in the diverticulum. In the former instance peptic ulcers have been found. Roentgenographically, the visualization of a Meckel's diverticulum is a rather infrequent occurrence.

CASE REPORT

The patient, a woman aged sixty, had had four previous operations, the first at the age of twenty for appendicitis, followed by operations for intestinal adhesions, ventral hernia and, finally, a hysterectomy.

Barium enema examination revealed an ileal diverticulum 18 inches from the ileocecal valve. It was 2.5 cm. long and 2 cm. at its widest diameter. It was symmetrical and pear shaped, with smooth borders, connected with the ileum by a neck 1.5 cm. long and 1 cm. wide (Fig. 1). Roentgenoscopically, its blind end was free and not tender. The diverticulum was removed and was found to have the same morphology as ileum. No aberrant tissue was found.

Roentgenographically, Meckel's diverticulum may be visualized by means of the oral fractional method or by barium enema. An additional roentgen method is to ex-

FIG. 1. The Meckel's diverticulum is seen in the right lower quadrant arising from the loop of ileum which is about 18 inches from the ileocecal valve on the roentgenogram.

* From the Division of Radiology of the Department of Hospitals, City of New York, Kings County Hospital, Dr. Richard A. Rendich, General Director.

amine the patient in the lateral upright position, when the Meckel's diverticulum would be seen anteriorly. The length of time the diverticulum remains visible depends on the size of the pouch, its position and the width of its neck.

CONCLUSION

A classical roentgen demonstration of a Meckel's diverticulum is presented.

114 East 54th St.,
New York, N. Y.

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A PRACTICAL TECHNIQUE FOR ROENTGEN PELVIMETRY WITH A NEW POSITIONING*

By A. E. COLCHER, M.D., *and* WALTER SUSSMAN, M.D.
PHILADELPHIA, PENNSYLVANIA

NUMEROUS techniques such as those by Jarcho, Thoms, Caldwell and Moloy, Snow, Weitzner, and others have been described. Each has given good results, but no one method has been found to be universally adopted nor embodies the following prerequisites:

1. Simple intersecting diameters of the true pelvis.
2. Simple positioning of the patient.
3. A simple ruler for rapid and direct mensurations comprehensible to the roentgenologist, obstetrician and general practitioner.

A method which fulfills these basic requirements is described in this paper.

I. INTERSECTING DIAMETERS OF THE PELVIS

We have divided the true pelvis into three levels with intersecting anteroposterior and lateral diameters. These levels include all the salient bony landmarks of the pelvis (Fig. 1, *C–D*).

Actual Inlet (First Level)

Lateral View. The actual inlet, as emphasized by us, starts from the inner upper margin of the symphysis pubis and extends along the iliopectineal lines through the level of the transverse diameter of the inlet and is projected to the sacrum (Fig. 2). The anteroposterior diameter of this strait is the line from *G* on the upper margin of the symphysis pubis to point *I* on the sacrum. Accordingly, on the lateral view, line *G–I* is drawn from the upper inner symphysis through the point *E* to the sacrum *I*. Point *E* is midway between the brim of the pelvis above, and the sciatic notches below; and about one-third the distance from *I* to *G*.

This we consider as the anteroposterior diameter of the actual inlet.

Anteroposterior View. The transverse diameter line is drawn at the widest points of the inlet *A–A'*.

In this manner, the anteroposterior diameter *G–I* intersects the transverse diameter *A–A'* on the same level at point *E*.

Mid-Pelvis (Second Level)

Lateral View. The mid-pelvis anteroposterior diameter is developed from the inner lower border of the symphysis, *P*, and projected through the mid-point between the spines of the ischium, *F*, to the inner aspect of the sacrum, *M* (Thoms[4]).

Anteroposterior View. The transverse diameter is measured between the spines of the ischium *B–B'*.

The above diameters intersect at point *F* and form the mid-pelvis diameters.

Outlet (Third Level)

Lateral View. The anteroposterior diameter is a line drawn from the tip of the sacrum *S* to the mid-tuberal diameter *T*. The bituberal points in this view are obtained by projecting straight lines from the lower border of the obturator foramen to the edge of the tuberosities of the ischium, *C–C'* (Snow[2]).

Anteroposterior View. The transverse diameter of the outlet is obtained by drawing a line from the lateral margin of the inlet, projected along the lateral wall of the forepelvis (noted as a dense white line on the roentgenogram) to the lower edge of the tuberosity of the ischium. With the same procedure on both sides, the transverse diameter *C–C'* is obtained (Snow[2]).

Thus, the anteroposterior diameter *S–T* intersects the transverse diameter *C–C'* at

* From the Departments of Radiology and Gynecology, Doctors Hospital, Philadelphia. Presented before the Philadelphia Roentgen Ray Society, May 6, 1943.

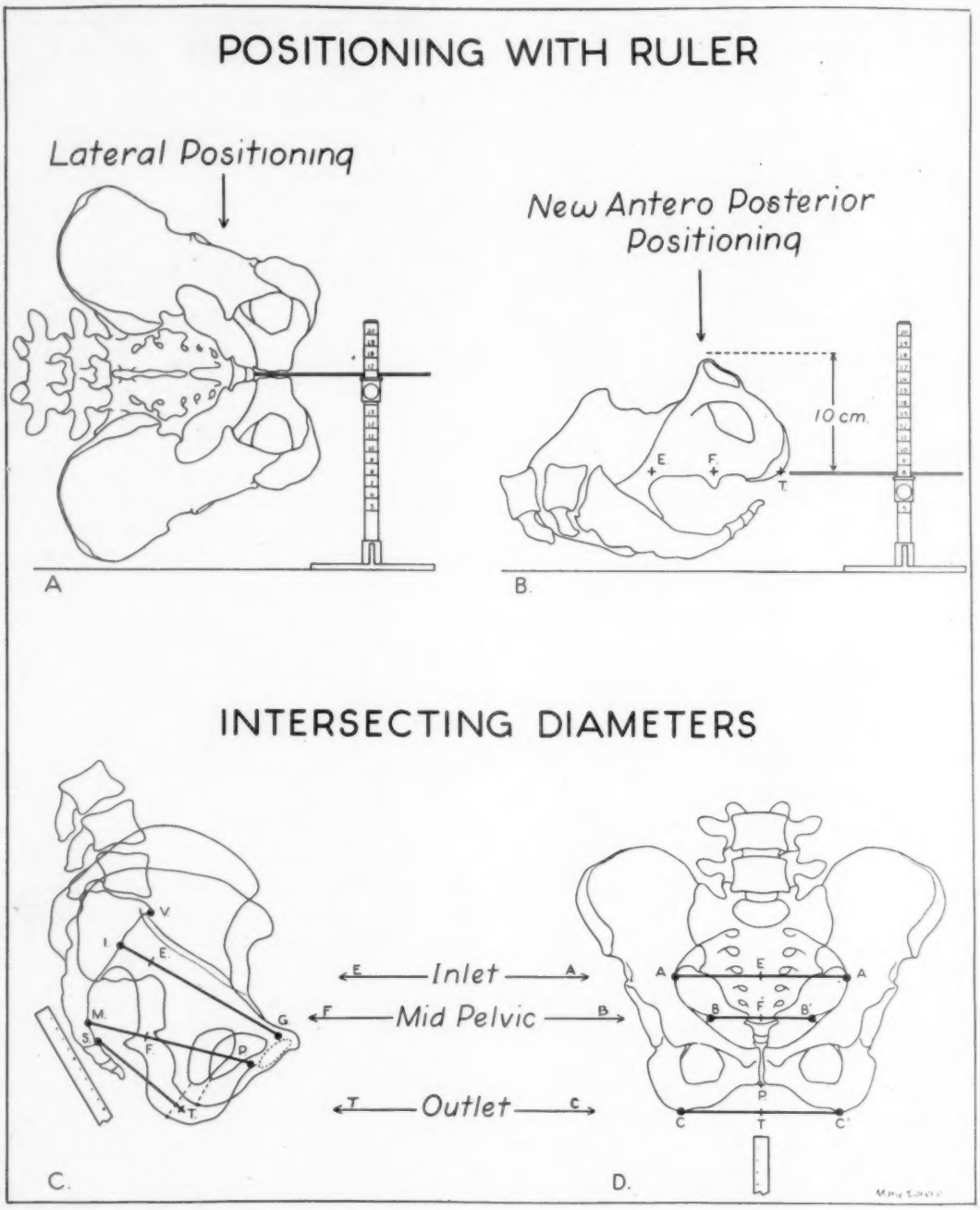

FIG. 1. *A* and *B*, diagrams showing lateral and anteroposterior positions with ruler. *C* and *D*, diagrams of the three pelvic levels with intersecting diameters.
Inlet: Actual anteroposterior diameter of inlet line *G–I* crosses through inlet transverse diameter *A–A'*.
Midpelvis: Anteroposterior diameter *P–M* crosses transverse diameter *B–B'*.
Outlet: Anteroposterior diameter of outlet *T–S* bisects midpoint of transverse diameter line *C–C'*.

the mid-tuberal point *T*.

True Conjugate. This is measured on the lateral view from a point between the upper inner aspect of the symphysis to the sacral promontory, *G–V*. It will be noted that there is no measurable transverse diameter on this level that would be of any value, since it would project into the false pelvis.

Therefore, the true conjugate is measured and considered as a separate entity.

Fetal Head

The ideal time for roentgen examination is at the beginning of labor or one week prior to the expected date of birth. The fetal head should be in the midline, otherwise fetal measurements are not considered accurate. We have taken two measurements of the fetal skull, the smallest and the greatest diameters in both the anteroposterior and lateral views. The average is recorded.

2. POSITIONING

Lateral View (Fig. 3*A*). This is the generally used true lateral exposure centered over the great trochanter. This results in a sagittal cross section through all the anteroposterior diameters of the pelvis. The fetal head, if in the midline, can also be measured (Granzow,[1] Weitzner,[6] Thoms[5]).

New Positioning for Anteroposterior View

Anteroposterior View (Fig. 3*B*). Since the lateral view just described has been conceded to be accurate, it now logically follows that any positioning in the anteroposterior exposure that would bring all the salient transverse diameters on the same level would result in an ideal setup for direct mensuration.

To meet this requirement, we have placed the patient flat on the back with the knees semiflexed and separated. This elevates the forepelvis and brings the bituberal diameter, bi-ischial diameter and the transverse diameter of the inlet practically on the same level and in the same plane as the table and film. Figure 4 shows a roentgenogram with diameters in lateral view. Figure 5 shows a roentgenogram with diameters in anteroposterior view.

3. ROTATING RULER*

A ruler devised by us (Fig. 1, *A* and *B*) consisting of an opaque metal strip with true centimeter scale perforations is used. This ruler is swiveled on a projecting arm attached to a vertical stem, which is also calibrated in centimeters. The stem is attached to a base parallel to the perforated scale. The ruler may be elevated, lowered and rotated. *Regardless of position, the scale is always parallel with the table top and the film.* Thus, when roentgenographed with the patient, the ruler will have the same distortion as the diameters on the same level. Therefore, the ruler markings on the film become the centimeter scale. These markings can be transferred to a sheet of paper, or a special chart to be described later, and used for the necessary measurements.

FIG. 2. Lateral roentgenogram of dried pelvis. Line *G–I* is actual anteroposterior diameter of inlet passing through the middle of the transverse diameter of the inlet as shown by lead markers.

Placement of Ruler

Lateral View (Fig. 1*A*). The ruler is placed at the mid-sacral spine parallel with the spine and film. Thus the centimeter rule markings are projected on the film for direct mensuration. It is simple and easy to accomplish.

Anteroposterior View (Fig. 1*B*). The ruler is placed at the level of the tuberosities of the ischium by direct manual palpation or

* May be obtained from Picker X-Ray Corporation.

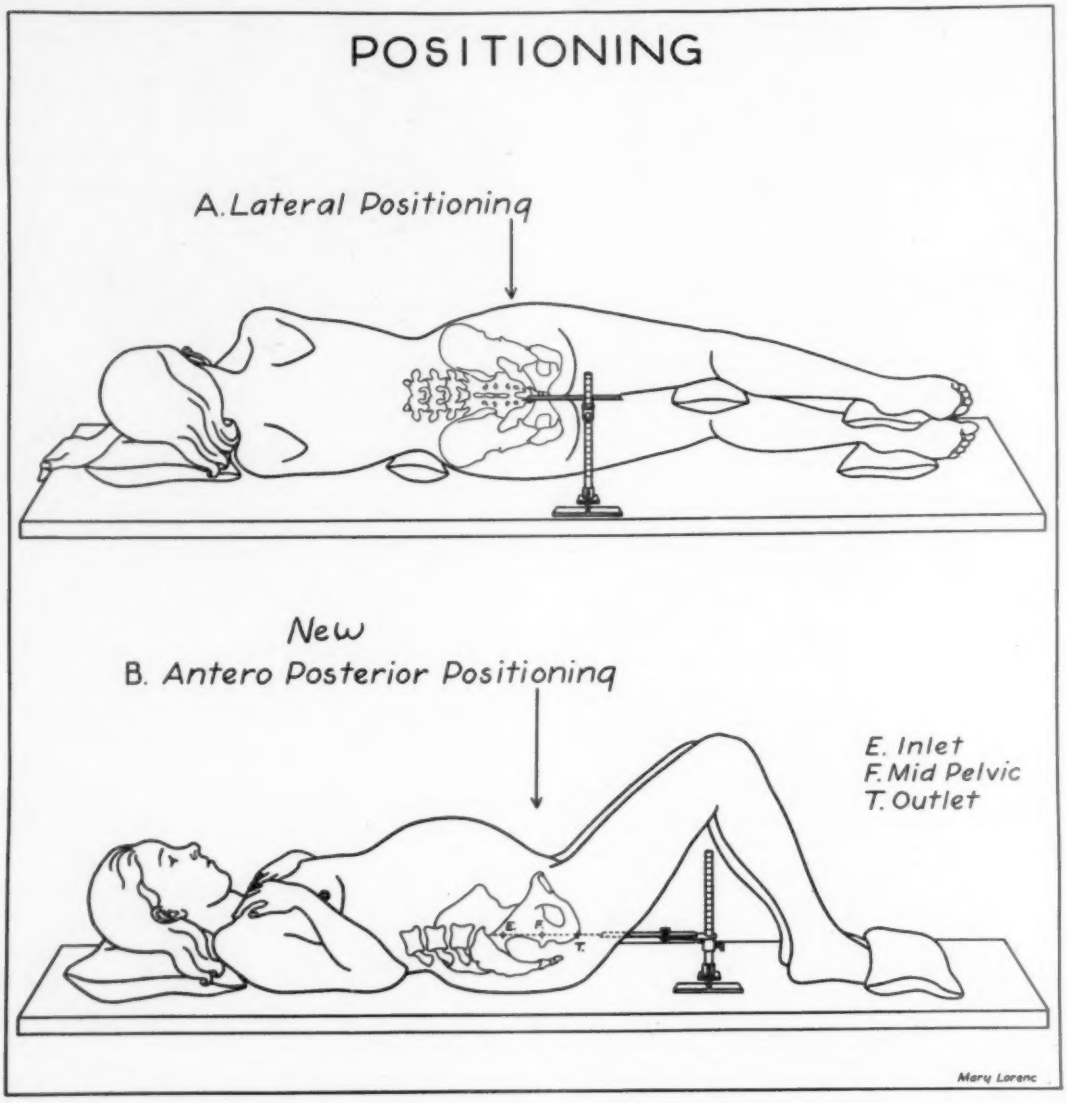

FIG. 3. *A*, lateral positioning. Patient on side; knee and thighs semiflexed. Ruler is at level of midsacra spine; the central ray is directed through the great trochanter of the femur any distance. *B*, new antero-posterior positioning. Patient flat on back; knees and thighs semiflexed and slightly separated. Ruler is placed at level with tuberosities of ischium. Central ray above symphysis at any distance.

lowering the ruler 10 cm. below the superior border of the symphysis pubis. The centimeter markings are projected on the film and have the same distortion as the transverse diameters of the pelvis.

The placement of the ruler 10 cm. below the top of the symphysis pubis is based on a study of 13 dried female pelves at the Wister Institute, 2 of our own, and living subjects. For soft tissues overlying the symphysis, 5 mm. were added. We have also dropped this ruler 8 and 12 cm. below the top of the symphysis pubis and the readings of the specimens did not vary more than 3 mm. to every 10 cm., or approximately 3 per cent variation. This includes the interspinous diameter which lies sometimes as much as 1.5 cm. below the level of the inlet and outlet.

Due to the proximity of the ruler to the table in this new position (about 4 inches) and with a 36 or 40 inch target distance, a slight

variation of the level of the transverse diameters of the pelvis becomes negligible in the final analysis.

The Chart (Fig. 6). It will be noted that the words lateral and anteroposterior are seen on the right and left margins of the chart. When the roentgenograms are ready, the chart edge marked lateral is placed on the ruler markings of the lateral view and the scale is marked off. A similar procedure is carried out with the anteroposterior view. The respective newly made rulers on the edges of the chart are used to measure the actual diameters on the roentgenograms.

EVALUATION OF DIAMETERS

The intersecting diameters of the actual inlet *G–I* and *A–A'* are added together. The total of 22 to 24 cm. is considered a normal range. The total of the mid-pelvis *M–P* and *B–B'* should average between 20 and 22 cm. Then the outlet *S–T* and *C–C'* are added; the normal range should average between 16 and 18.5 cm. (Snow[3]).

Fig. 5. Lateral roentgenogram (as in diagram of Fig. 1*C*). Line *G–V* is textbook true conjugate; *G–I*, anteroposterior diameter of actual inlet; *P–M*, anteroposterior diameter of mid-pelvis; *T–S*, anteroposterior diameter of outlet. Centimeter ruler markings on roentgenogram used for measurements.

Fig. 4. Anteroposterior roentgenogram (as in diagram of Fig. 1*D*). *A–A'*, transverse diameter of inlet; *B–B'*, transverse diameter of mid-pelvis at the level of the ischial spines; *C–C'*, transverse diameter of outlet at level of tuberosities of ischium. Centimeter ruler markings on roentgenogram used for measurements.

The textbook true conjugate is evaluated on its own measurements as a separate diameter for obstetrical consideration. The normal range is 11 to 11.5 cm.

The fetal head measurements can be evaluated by the general average in both the anteroposterior and lateral views.

Consideration of the different pelvic diameters with the various measurements of the fetal head, such as the biparietal and the suboccipitobregmatic diameters, should also receive separate obstetrical evaluation. The latter diameters are easily measured when the fetal head lies in the anteroposterior or transverse position. The fetal head must be in the midline for accuracy.

SUMMARY

1. A simple positioning of the patient for roentgen pelvimetry has been described.

ANTEROPOSTERIOR VIEW — CHART — LATERAL VIEW

Centimeter Scale Taken From Film

TRUE CONJUGATE G - V

FETAL HEAD: Anteroposterior View; Lateral View; Average

		Diameters	TOTAL	NORMAL RANGE OF TOTAL
ACTUAL INLET	Anteroposterior	G - I		22 - 24
	Transverse	A - A'		
MID-PELVIS	Anteroposterior	P - M		20 - 22
	Bispinous	B - B'		
OUTLET	Anteroposterior	T - S		16 - 18.5
	Bituberal	C - C'		

Type of Pelvis — Lumbo-sacral articulation

Position of Fetal Head — Separation of Symphysis

Moulding of Fetal Head — Position of Coccyx

Remarks:

FIG. 6. Sample chart. Ruler markings at sides taken from roentgenograms used as final ruler for measurements and records of pelvic diameters and fetal skull.

2. Only two exposures are required, an anteroposterior and a lateral view, taken at any distance.

3. The ruler gives direct centimeter measurements on each film without need of correction tables.

4. The true conjugate is considered as a separate entity since it has no transverse diameter in the true pelvis on the same plane.

5. The diameters of the pelvis—namely, those of the inlet, the mid-pelvis and the outlet—are measured along intersecting lines embracing all bony landmarks.

6. The fetal head, when presenting in the midline, can be measured in both the lateral and anteroposterior views.

7. A quick and accurate method for measuring the subpubic angle by measure of a precalculated table (Fig. 7) is included.

Spruce Street Medical Building
Philadelphia, Pennsylvania

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DISCUSSION

DR. PAUL C. SWENSON, Philadelphia. Dr. Colcher and Dr. Sussman have added another relatively simple, easy method of pevimetry to the growing list of variations of one method

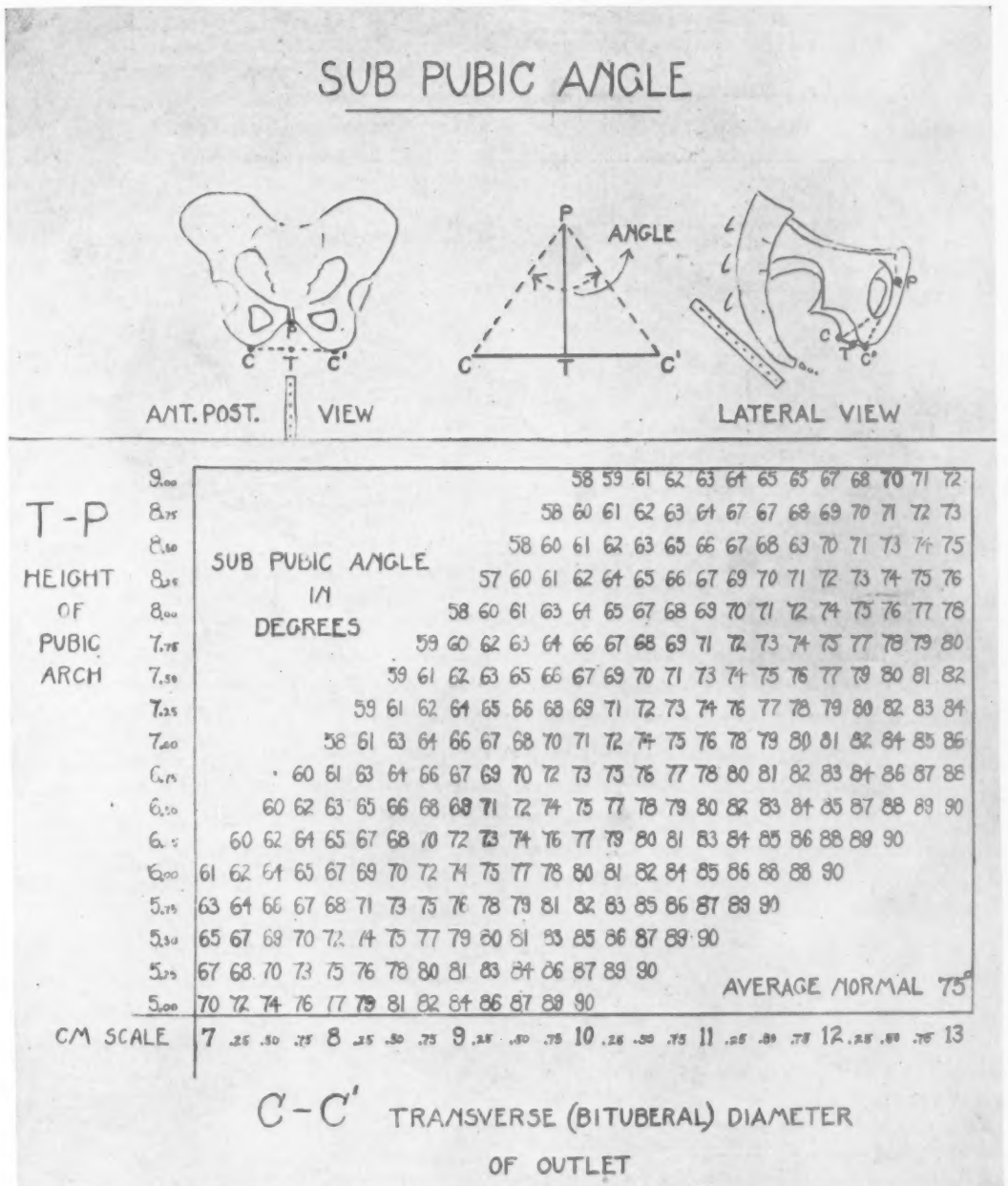

FIG. 7. A new method with a precalculated table for accurately measuring the subpubic angle. May be used with other techniques. The height of the subpubic arch is measured on the lateral view from the mid-tuberal point, *T*, to the lower inner symphysis, *P*. The base line of the subpubic arch, *C–C'*, is measured on the anteroposterior view. Where these two measurements meet in the table is the subpubic angle in degrees.

or another. I am impressed with its practicability.

I think it remains for every office or department of roentgenology to stick to one method that has proved practical in its experience and learn to use it well, being always mindful of the dangers that might be forthcoming from putting too much reliance on measurements alone. Exact measurements are quite all right, provided the data obtained are properly evaluated. A proper clinical approach is always necessary.

I would like to say a word about the figure given by Dr. Snow and quoted by the authors relative to the number of cases of serious arrest of labor. Eight to 10 per cent is given as the usual figure by several authors in cases of arrest due to cephalopelvic disproportion. This, I think, should be broken down a little in order that it be not misunderstood. For example, at the present time, the results at the Sloane Hospital show cases of serious arrest broken down into a cesarean section rate of 4.4 per cent and a 5.0 per cent rate of mid-forceps operation, making a total of 9.4 per cent. Of this 4.4 per cent of cesarean section, about 2 per cent is due to absolute disproportion which can be recognized without the use of measurements at all. One glance at a roentgenogram corroborating an already suspected disproportion clinically, will tell one that section is indicated. The other 2.4 per cent will be border line and require careful analysis based on the amount of disproportion, the type, and the pelvic morphology. An additional 5 per cent of cases include the use of mid-forceps. In these cases a disproportion exists which, with the help of forceps, will make the delivery from below relatively easy. It is in this group where a knowledge of the pelvic type, as well as the border line disproportion is important. The border line disproportion becomes increasingly important as the dangers inherent in a bad pelvic type are found to be present. Moreover, we must always keep in mind the varying extra-osseous factors which may compensate for minor disproportion. A border line situation is a more important factor in a forty year old primipara than in a young multiparous patient with no history of dystocia.

The point I wish to make is that in this total of 9.4 per cent of serious arrests, about 7 per cent will be a problem of careful correlation of morphology and size, and, it is in this group where the roentgen examination can be of real value *but* it is not going to be a matter of 2 mm. one way or the other as regards size of head and pelvis.

If we as roentgenological consultants are to keep the clinician interested in the diagnostic advantages of roentgenography in obstetrics, we will have to combat any tendency to regard its use as a rule of thumb procedure and instead attempt to emphasize its broader aspects, which combines a knowledge of the accurate pelvic diameters with the other known factors, both roentgenologic and clinical. If we do not do this, any procedure we popularize will easily lead us into error and place the roentgenologic aid in disrepute. I mention this only to emphasize what Dr. Colcher has already said.

The authors' method of getting the greatest transverse diameter and the bituberous on the same plane is very ingenious and they are to be congratulated on having worked this out. However, recent work has emphasized the advantages of pelvimetry in the erect position, both lateral and anteroposterior. This is particularly good for determining whether the head dips into the inlet. At least the lateral view will probably be desirable and it would be of advantage if the method under discussion could in some way be applied to this view. Otherwise, in my opinion, it should be added to the other roentgenograms obtained.

Dr. Colcher has emphasized the fact that the real level of the pelvic inlet is below what has always been considered the true conjugate. This we have recognized for some time and I am glad he has emphasized it.

Mention was made of the ease of pelvic typing when certain measurements are available. While this is quite true, we, in our department, have found that nothing quite supplants the ability which the stereoroentgenogram allows to study the pelvic image as a composite whole. It goes without saying, even though I had a part in developing the stereoscopic method, that the added expense which the method entails makes it rather expensive for the average office, unless the volume of work is large enough to warrant its use, and unless the personnel becomes experienced in the stereoscopic technique of measurement.

Again let me commend the essayists for their contribution. The method is apparently practical and I hope to be able to give it a thorough trial.

DR. HARRY D. EVANS, Philadelphia. I have been partial to Snow and Lewis' method, for in my hands I have found it to be accurate and not too complicated. Recently, through the courtesy of Dr. Colcher, I have had the opportunity to use his ruler in 6 or 7 cases upon which I compared the results with those of measurements by Thoms' technique and also that of Snow and Lewis. I am happy to report that the greatest discrepancy noted between Dr. Colcher's method and the other techniques mentioned was never in excess of 3 mm. When we consider that errors up to and including 5 mm. are universally accepted in this kind of work, the results emphasize the accuracy claimed by Dr. Colcher.

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CLEIDOCRANIAL DYSOSTOSIS*

By C. G. LYONS, M.D.
Veterans Administration Facility
WEST LOS ANGELES, CALIFORNIA
and
J. G. SAWYER, M.D.
Veterans Administration Facility
BILOXI, MISSISSIPPI

CLEIDOCRANIAL dysostosis is a comparatively rare congenital defect of the bony skeleton, less than a hundred cases having been reported in the literature thus far. It is a syndrome in which the chief characteristic finding is a complete or partial absence of one or both clavicles. As so often happens, when one congenital defect is found in the body, another may also be found. In this condition one of the more constant defects accompanying that of the clavicles is a variation in the development of the skull bones.

Morand, in 1766, first described a case, which is not considered typical. In 1857, Stahmann reported a case of congenital absence of the left clavicle in a boy aged nine years. Barlow in 1883 reported a case of congenital absence of both clavicles and malformation of the cranium in a girl aged two years, probably the first typical case described. In 1897 Marie and Sainton reported 4 cases and applied the term "hereditary cleidocranial dysostosis" to the anomaly syndrome. Fitchet[2] in 1929 made a complete survey of all reported cases in the literature, summarizing each report. He stated that the chief features of this condition, as listed by Abrams from Marie and Sainton's article, are grouped under four headings as follows: aplasia, more or less marked, of the clavicles; exaggerated development of the transverse diameter of the cranium; delay in the ossification of the fontanelles; hereditary transmission.

There is much variation in the occurrence and degree of changes under the above headings, every case recorded not demonstrating all these findings. The anomalous development of one or both clavicles is the really characteristic defect. Therefore, Fitchet believed the term "hereditary cleidocranial dysostosis" should be discontinued and "congenital cleidal dysostosis" substituted instead, since the condition may be either hereditary or non-hereditary, with or without other anomalies. However, common usage will probably continue the designation of this condition by the older terminology.

The clavicular deformity may vary from defects at the acromial end to complete absence of one or both clavicles. The latter finding is very unusual, however. Most frequently, it is the middle portion of the clavicle that is involved, and the defect may vary from that of a narrowing to a thin bony or cartilaginous bar to the presence of only a ligamentous structure.

The skull defects may consist of brachycephalic changes, prominence of the frontal and parietal bosses or delay in closure of sutures and fontanelles. In 1907, Hultkrantz, making a survey of all cases reported up until that time, summarized by Fitchet, described the most characteristic skull finding to be the disturbed suture formation. These disturbances were explained to cause most of the other skull deformities. Kelley[3] described the skull as showing a globular, distended cranial vault with a bulbous forehead. He noted that the face is small in many of these cases, and that the suborbital region is depressed and sunken.

Regarding the familial and hereditary aspects, Marie and Sainton, according to Fitchet, stated that the condition had never

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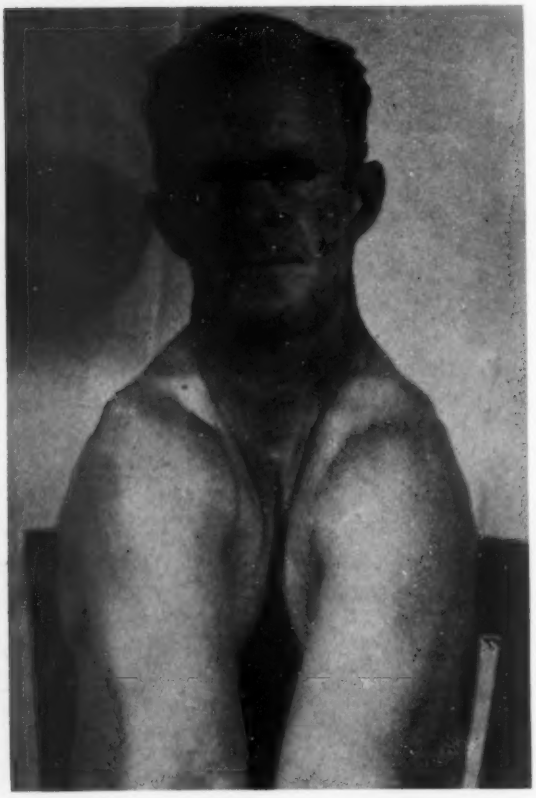

FIG. 1. Approximation of shoulders to midline anteriorly.

been known to run more than two generations, there being a tendency to revert to the normal type. However, later investigators have reported cases to extend through three and even four successive generations. There are also many cases reported that give no familial or hereditary history. On the basis of this fact, Fitchet divided the cases into two types: those with a familial and hereditary history and those without. Probably more of the cases come under the head of those giving no familial or hereditary history.

There have been other changes accompanying the clavicular deformity reported in the literature. Most of the patients are described as being of small stature. Anomalies of dentition are common, consisting of delay in the appearance of permanent teeth, incompleteness or irregularity of the second set of teeth, a proneness to caries, missing teeth or supernumerary teeth. A high palatal arch is also quite frequent. Defects in the bones of the hands and feet, including shortening of the toes, have also been reported.

The following case is being reported as meeting most of the requirements for classification as cleidocranial dysostosis, as set down by various authors in the literature.

REPORT OF CASE

T. H., male, aged forty-four, a veteran of World War I, was noted by routine physical examination and chest roentgenography at the Veterans Administration Facility at Biloxi, Mississippi, to have a deformity of the clavicles. Besides the anomalous findings, chronic bronchitis and a psychopathic personality with pathological emotionality were evident. There was no history or evidence of venereal disease.

The patient said that he knew from childhood of his clavicular deformity. It was discovered by Army physicians at the time of physical examination prior to his war service. As a consequence, he was placed on limited duty in the non-combat section and served from August 30,

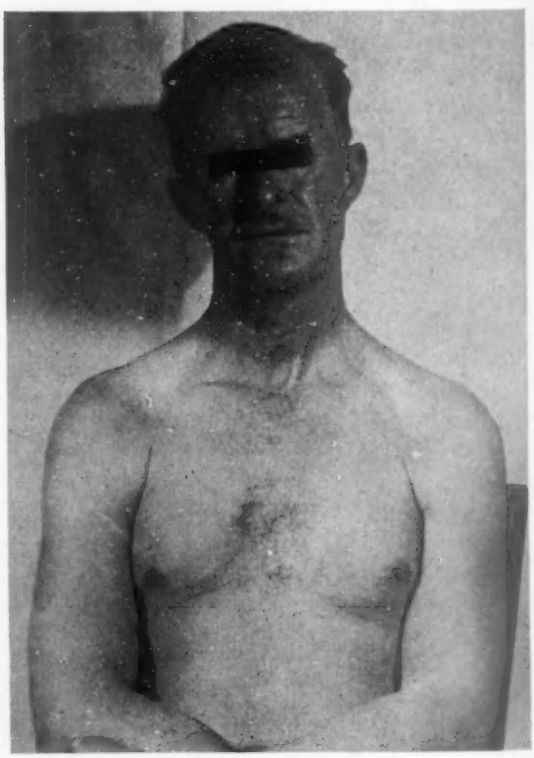

FIG. 2. Normal position of shoulders.

1918, to January 22, 1919, as a private in a developmental battalion.

Questioning as to a hereditary or familial history of this defect revealed that he had a brother, two years older than he, who had an absence of the left clavicle. This brother died in 1925. He recalled no similar defect in any other member of his family. The remaining family history and past personal history revealed nothing remarkable.

With respect to the clavicular deformity, it was noted on physical examination that he

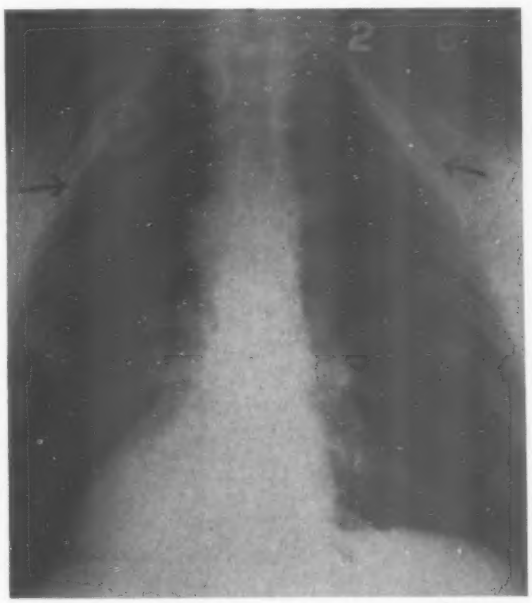

Fig. 3. Arrows point to rudimentary clavicles. Evidence of chronic bronchitis is also displayed.

carried his shoulders forward, like one who has suffered a bilateral clavicular fracture. Examinations of the clavicles revealed only a small, thin nubbin of bone, extending outward from the sternum on either side, each measuring about 1 cm. in width and 7 cm. in length. These pieces of bone were freely movable, and manipulation caused no discomfort. The shoulders were extremely mobile, much more than normal, and could be made to approach the midline anteriorly until they were only 5 to 6 cm. apart. The entire upper thorax was conical in shape. The muscles of the shoulder girdle appeared very well developed. He had a large head, globular in form, with prominence in the frontal and parietal regions. The face appeared small, in comparison with the remainder of the head.

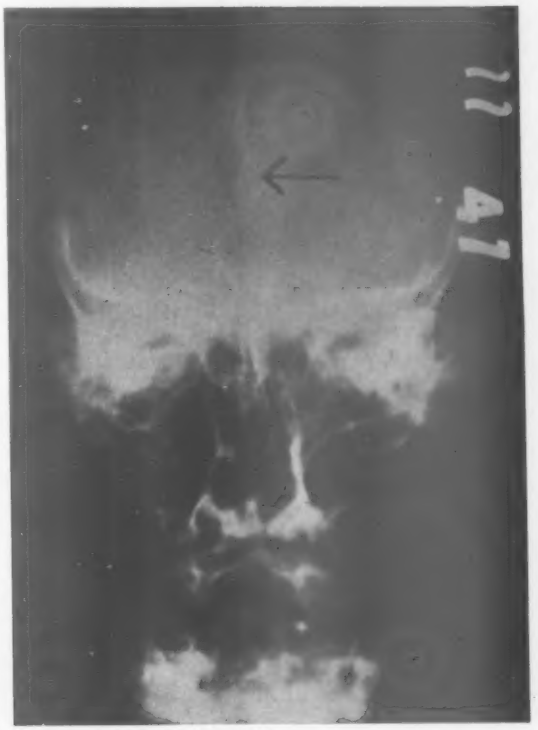

Fig. 4. Metopic suture line.

There was a depression in the malar region on both sides. All the teeth were missing. He gave

Fig. 5. Fingers, left hand, showing shortening of middle and distal phalanges and foreign body embedded in proximal phalanx, index finger.

FIG. 6. Shows marked shortening of middle and distal phalanges of all toes.

a history of having had very poor teeth for years before they were extracted in 1930. He said that at the time of total extraction four supernumerary teeth were discovered in the upper jaw and were removed. He had a high, markedly large palate. It was further noted that the fingers of both hands appeared short and stubby and that the feet were small and the toes short. He said he wore size $4\frac{1}{2}$ shoes. His height was 67 inches and his weight 141 pounds. His whole stature appeared somewhat slight.

Roentgen examination of the chest revealed a congenital deformity of the clavicle bilaterally. All that could be seen of the clavicles was a small bony nubbin extending outward from either side of the sternum in what would normally have been the position of the clavicles. These bony shadows measured approximately 7 cm. long and 1 cm. wide. The structure of the bone appeared normal. The remainder of the clavicles was entirely absent. The lung fields displayed evidence of a chronic bronchitis. Roentgenograms of the skull showed a somewhat globular shape of the cranial vault and a persistent frontal or metopic suture. The facial portion appeared small in relation to the remainder of the skull. The roentgenograms of the hands displayed a shortening of the middle and distal phalanges bilaterally and evidence of a foreign body within the proximal phalanx of the fingers of the left hand. Roentgen examination of the feet displayed a marked diminution in size of the distal and middle phalanges, especially the distal ones of the first toes, which appeared to be only about one-half normal size.

A roentgenogram taken of the right mandible following complaint of a small, tender swelling along the lower border of this bone revealed the presence of a supernumerary tooth in the anterior molar region. The tooth appeared to be embedded within a dentigerous cyst, and it was noted that the root of this tooth appeared to protrude slightly below the lower border of the mandible.

The patient also complained that it was difficult for him to sleep on his side. He stated that the upper shoulder would fall forward and impede his breathing. He was unable to lie on his back comfortably because his bronchial condition would cause him to cough. He therefore wore a special type clavicular brace which held his shoulders back and afforded him more comfort.

FIG. 7. Right mandible, showing supernumerary tooth embedded in dentigerous cyst with its root protruding below the lower margin of the bone.

The outstanding clavicular deformity, accompanied by a definite familial history and the presence of dental defects, skull defects and deformity of the hands and feet, seems definitely to warrant a diagnosis of so-called cleidocranial dysostosis.

The authors are indebted to Mr. S. C. Hemmings for the reproductions in this report.

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REGRESSION OF BONE METASTASES FROM BREAST CANCER AFTER OVARIAN STERILIZATION*

By MAX RITVO, M.D., *and* OSCAR S. PETERSON, JR., M.D.
BOSTON, MASSACHUSETTS

AN EXTENSIVE literature has accumulated in recent years stressing the importance of ovarian sterilization in the treatment of metastatic lesions from cancer of the breast.[2,3,5,11,14,15,21,22] Local recurrences and deep, visceral metastases generally seem to show little or no response after radiation treatment directed to the ovaries, although occasionally very striking benefits in cases of these types are recorded in the literature.[4,7,8] However, the most satisfactory results seem to occur in patients with osseous metastases and several instances in which very definite and marked improvement of metastatic lesions had taken place have come to our attention. The purpose of this communication is to stress again the value of this form of therapy and to record a series of cases from the Pondville Hospital which have benefited from roentgen ovarian sterilization.

The rationale of the method is well established and the opinion of many authors is that, in properly selected cases, there is definite value in this mode of therapy.[6,12] The absence or withdrawal of the ovarian hormone is apparently the important factor in producing the favorable results.[10,13,18,20] In many instances, roentgen treatment directed to the pelvis with consequent sterilization has resulted in partial or complete recalcification of metastatic bone foci far removed from the area exposed to irradiation with no therapy directed to the local lesions. The question as to whether this form of treatment results in appreciable prolongation of life is still in doubt and cannot at present be definitely answered in the affirmative as the evidence is extremely difficult to evaluate and the conclusions of different authors are not in agreement.[9,16] It is our belief that there has been unquestionable prolongation of life in some instances, while in other cases there has apparently been no appreciable improvement in this respect.

There are, however, certain definite benefits which may be expected in properly selected cases. The chief among these can be summarized briefly as follows: (1) there is usually prompt and effective relief of pain; this may be of considerable duration and frequently lessens or temporarily obviates the need for morphia and other habit forming narcotics; in some instances, roentgen therapy abolishes pain when sedatives have failed to give relief; (2) the lesions in the bones regress partially and in some instances entirely reossify to disappear completely; (3) fractures with their resultant deformities and prolonged disabilities may be prevented; (4) there is great improvement in the patient's general condition with increased appetite and gain in weight; (5) there is marked prolongation of the patient's usefulness, many being able to continue their regular occupations for considerable periods with consequent benefit not only to the individual but to the family and the community; (6) there is striking betterment of the patient's mental status with helpfulness and optimism replacing the feeling of hopelessness and despair which so commonly occurs.

The technique of roentgen sterilization varies in its details in various clinics, but in general follows fairly well established rules. The object is to produce the desired result without injury to the skin and with as little disturbance of the patient through roentgen sickness as possible. In our clinic, the total dosage is usually 2,000 to 3,600 r, varying with the size of the patient. Two to four fields, each measuring about 15 by

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15 cm., are used over the lower portions of the abdomen and lower back, the rays being directed into the pelvis. The filtration is 0.5 mm. of copper and 1.0 mm. of aluminum, giving a half-value layer of 1.0 mm. of copper. Two hundred kilovolts is the usual voltage and the target-skin distance is 50 cm. One field is exposed daily in rotation and the customary single dose is 200 r, although it may safely be increased to 250 or 300 r. With the larger dosages, a permanent sterilization usually results and in most instances is followed by hot flashes and other symptoms which may accompany the menopause. In earlier cases, total doses of only 1,200 to 1,800 r were administered with apparently satisfactory results. With smaller amounts of radiation the periods may return after an interval of six to eighteen months, which is undesirable and may necessitate another course of treatment. With our present therapy, there is little or no general reaction to the treatment and no permanent skin damage results. Occasionally there is nausea, malaise, loss of appetite or diarrhea, in which instance treatment may have to be suspended for a few days. Forcing fluids, giving large amounts of fruit juices and the administration of vitamin B complex usually control the untoward effects.

Some authors have favored routine ovarian sterilization of all patients with cancer of the breast in the premenopausal age periods.[17,19] It is our belief that in the light of present day knowledge, this is not advisable. Ahlbom,[1] in a very detailed study, showed that no definite benefits either as regards prolongation of life or the lessening of the incidence of metastatic lesions occur after ovarian sterilization. Moreover, patients suffering from cancer of the breast, particularly those who have been operated upon, should not have the added burden of a premature menopause unless it is reasonably certain that definite benefit is to be expected from the procedure. It is our experience that while ovarian sterilization has been followed by regression or total disappearance of osseous metastases, it has not prevented new metastatic lesions from appearing. In 3 of our cases, discussed in greater detail below, metastases in the bones have disappeared after roentgen irradiation of the pelvis only at a subsequent date to have new areas of destruction reappear in different portions of the skeletal system.

It is our usual procedure to observe all patients with cancer of the breast carefully at frequent intervals after operation or radiation therapy. Roentgen search for metastatic foci is carried out routinely two or three times annually. This usually comprises roentgenograms of the chest, skull, spine, pelvis and upper femurs. Roentgen examinations of the other bones are also made if there is pain, swelling or other clinical indication. At the first evidence of the development of bone metastases, roentgen sterilization should be performed. Roentgen therapy directed to the involved areas may be administered simultaneously in an attempt to speed the regression of the lesions, although in some instances the metastases regress or disappear without such local therapy. The general consensus is that about one-third or more of the cases treated may be expected to benefit from this form of treatment. We have not attempted to present statistics on our cases as, in many patients, the disease was so far advanced at the time treatment was instituted that death ensued too soon to permit of proper evaluation. In numerous instances, however, the benefits after sterilization were so striking that, though only an occasional case responded favorably, the treatment would nevertheless be very much worth while. There are no contraindications to ovarian sterilization and it may be used whenever there is any reasonable hope of benefit. Since pregnancy is contraindicated in patients with metastatic lesions, this is an additional benefit incidental to sterilization. Patients who are markedly cachectic should receive smaller daily doses and have the treatments distributed over a longer period than those in good physical condition.

CASE REPORTS

Case I. M. E. G., female, white, aged thirty-five. In April, 1937, she noted slight retraction of the left nipple and three months later felt a lump in the breast. She first sought medical attention in October, 1937, at which time the tumor had increased considerably in size. On October 25, 1937, left radical mastectomy was performed. Pathologic report was carcinoma simplex with metastases in four of seven nodes. The following month roentgen therapy was administered, 1,500 r to the left supraclavicular area, the left axilla anteriorly, the posterior aspect of the left axilla, and the left chest anteriorly.

In November, 1938, there was no evidence of local recurrence. A mass was palpable in the right breast. The patient complained of backache, and roentgen studies revealed extensive metastatic disease in the ribs, spine, pelvic bones and upper femurs. Roentgen therapy was administered, 1,200 r to the pelvis anteriorly, 900 r to the right side of the pelvis posteriorly and a similar amount to the left side posteriorly. In January, 1939, she stated that she had had prompt relief of pain and was entirely symptom free; her last menstrual period had been on December 12, 1938. In August, 1939, she reported feeling entirely well and that there had been a gain of 18 pounds in weight since the last roentgen treatments.

Roentgen studies carried out February, 1940, showed filling in of the disseminated osteolytic metastases. The patient looked well and reported she had no complaints. In August, 1941, she stated that she had been symptom free until a very short while previously, although recently she had noticed increasing apathy, weakness and weight loss. On physical examination, there was evidence of marked cachexia and the picture was consistent with advanced generalized carcinomatosis. There was no local recurrence. The patient died on August 19, 1941, in uremia. Permission for postmortem was not obtained.

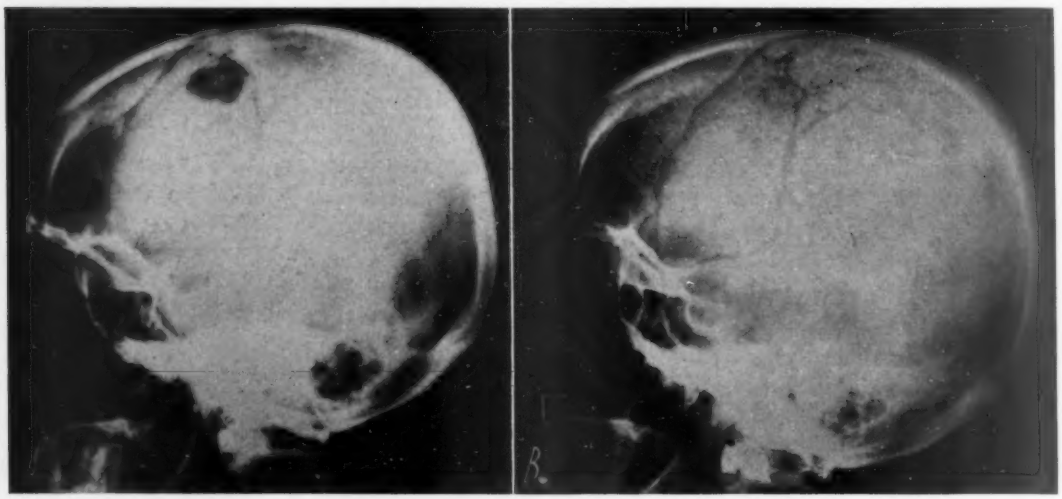

Fig. 1. Case II. *A*, roentgenogram of the skull, July 1, 1933, showing large, rounded area of radiance in the parietal region. Metastatic carcinoma. *B*, June 23, 1937, almost four years later. The metastatic focus in the parietal region seen in *A* is now evidenced by an area of slightly mottled density, having disappeared almost completely.

Case II. M. B., female, white, aged thirty-seven, married. In February, 1932, the patient felt tenderness in the right breast and a lump about the size of a walnut. In May, 1933, she showed advanced malignant disease involving the right breast with large masses of metastatic lymph nodes in the right axilla. She was treated with radium at that time, receiving 6,720 milligram-hours, with sixteen needles of 2 to 3 mg. each, distributed throughout the breast and an additional dose of 3,360 mg-hr. inserted directly into the main mass in the breast. Roentgen studies on July 1, 1933, revealed metastatic cancer involving the skull with an area of destruction measuring about 2 cm. in diameter in the parietal region on the left side (Fig. 1*A*).

In October, 1933, sterilization by the roent-

gen method was performed, the dosage being 600 r to an anterior and 600 r to a posterior pelvic field, each 10 by 15 cm., with 200 kv. and 0.5 mm. copper filtration; 1,200 r was also delivered to the right chest anteriorly. The patient was observed at intervals thereafter and the primary disease apparently remained under control. The lesion in the skull, described above, filled in completely and roentgen studies on March 12, 1936, and again on June 23, 1937, showed no evidence of recurrence in the skull was no tenderness or weight loss. Roentgen studies again were negative.

The patient was seen periodically about three times annually and in July, 1940, metastatic disease was demonstrated in the ribs, left ilium, skull and lungs (Fig. 2*A*). In October, 1940, roentgen sterilization was performed, the dosage consisting of 400 r each to right and left anterior pelvic fields and 600 r each to the right and left posterior pelvis. The factors used were 200 kv., 0.5 mm. copper filtration and 10 by 15 cm.

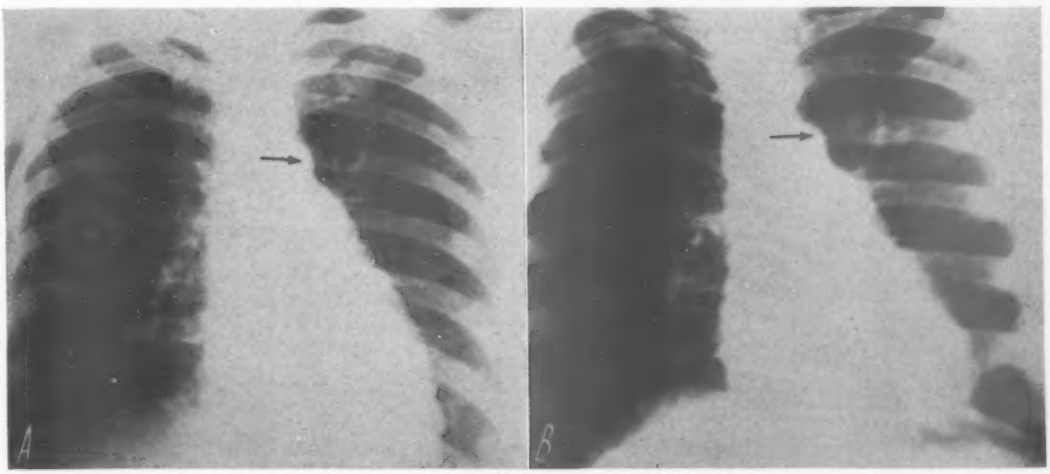

Fig. 2. Case III. *A*, July 13, 1940. There is a metastatic lesion in the posterior aspect of the sixth left rib with a large rounded area of destruction in the paravertebral portion of the rib. *B*, same patient nearly two years later on June 29, 1942. The lesion in the sixth left rib has reossified almost completely.

(Fig. 1*B*). In August, 1936, she began to have left frontal headaches and failing vision in the left eye. Roentgen therapy to the head, left frontal area, was administered in July, 1937, 1,200 r being given with 200 kv. and 0.5 mm. copper filtration. The patient died on February 10, 1939, approximately seven years after her first observation of the mass in the breast, of disseminated metastatic cancer.

Case III. L. A. R., aged thirty-nine, married. The patient noticed a lump in the right breast in 1935 and visited her physician but was not referred for surgery till June, 1936, at which time a radical mastectomy was performed. The pathologic report was adenocarcinoma with axillary metastases. One year later, there was no evidence of local recurrence. A node was palpable in the left axilla. The patient complained of pain between the scapulae. Roentgen studies of the chest, skull, spine and pelvis revealed no evidence of metastases. In March, 1938, she still complained of back pain; there fields. Three months later, the patient was entirely symptom free except for frequent hot flashes. The metastatic area in the pelvis had filled in; the lesion in the skull was definitely smaller, and the destructive process in the ribs was less marked than previously. In February, 1941, the patient received 1,600 r over the right chest. When seen a few months later, she was symptom free. In April, 1942, the metastatic areas in the skull, ribs and pelvis had disappeared almost completely and the patient looked and felt well (Fig. 2*B*).

Case IV. R. L. V., female, white, aged forty-one, married. In 1928, she first noted the appearance of a small lump in the left breast and the following year a left radical mastectomy was performed. No report of the microscopic findings could be obtained. There was wound sepsis and abscess formation after this operation. In 1931, she was operated on for recurrent tumor, the pathologic report of which was as follows: "a mass of glands measuring 4 by 2 cm. showing

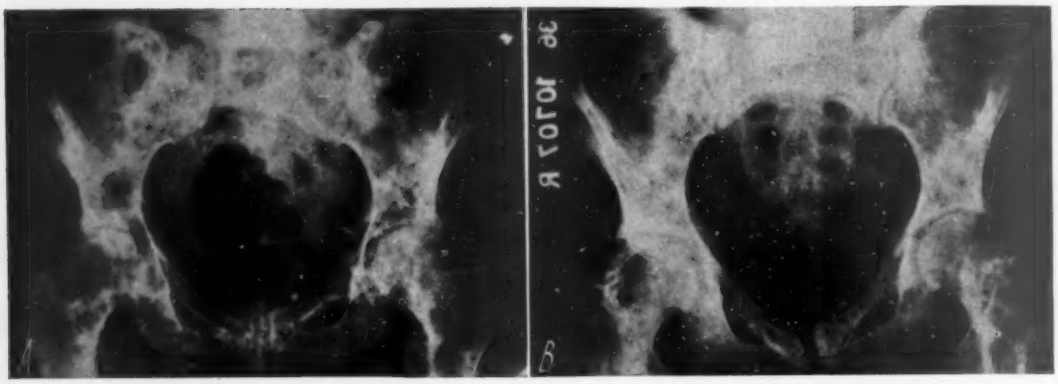

Fig. 3. Case v. *A*, April 15, 1936. There are advanced osteolytic changes involving the lumbosacral region, the bones of the pelvis and the upper femurs. *B*, September 9, 1936. There has been extensive proliferation of new bone with marked regression of the metastatic lesions.

metastatic carcinoma," suggesting that the previous operation had been incomplete. Three years later, a mass about 3 cm. in diameter recurred in the mid-portion of the operative scar and this was removed with the high frequency knife. The wound was not closed and did not heal. Seven months later, a large ulcerative tumor about the size of an egg was removed in the region of the previous excision. The pathologic report was "rapidly growing adenocarcinoma."

The patient entered Pondville Hospital in September, 1935, with recurrent carcinoma involving the left chest wall in the region of the operative wound and left axilla. A biopsy was performed, the report being returned as carcinoma simplex. Roentgen studies revealed early metastatic disease with extensive destruction of the body of the left scapula, the upper third of the shaft of the left humerus, and the ribs. The patient was given roentgen therapy, 1,200 r being administered to the front and back of the left chest. Sterilization was performed in October, 1935, 600 r being delivered to the anterior pelvis and a similar amount to the pelvis posteriorly. The kilovoltage was 200, with filtration of 0.5 mm. of copper and 1 mm. of aluminum over a field 15 by 15 cm. In November, 1935, a total dose of 900 r was delivered to the left chest anteriorly. The patient was seen a month later and felt well. There were no palpable masses. The skin in the left axilla showed extensive erosion.

In July, 1936, she returned for observation. A firm mass was palpable in the right breast and there were numerous enlarged lymph nodes in the right axilla. The metastatic lesions in the bones, above described, showed practically complete regression with extensive new bone formation in the humerus, scapula, and ribs on the left, these areas now appearing practically normal on the roentgenogram. The patient showed evidence of much weight loss. Death ensued on September 27, 1936. Permission for autopsy was not obtained.

Case v. M. L. B., female, aged forty-three, married, no pregnancies. Her menstrual history had been essentially normal, although the periods had been slightly irregular and the flow had decreased slightly during the past year. In October, 1933, she first discovered a lump in the right breast and three days later the breast was removed. Six roentgen treatments were given postoperatively and a few months later three more roentgen treatments were administered. Two years later, she began suffering from pain in the back which became progressively more severe. In April, 1936, she entered Pondville Hospital. There was no local recurrence on the right, but a neoplasm was present in the left breast. Roentgen studies revealed extensive skeletal metastases with multiple destructive lesions in the skull, spine, ribs, pelvis and femurs (Fig. 3*A*). Sterilization was performed. The dose given was 900 r to an anterior and posterior pelvic field with 200 kv. and 0.5 mm. copper filtration. In June, 1936, she was quite comfortable and was able to sit up. Roentgen studies on September 9, 1936, revealed marked improvement of the bone lesions with extensive filling in and much new bone formation (Fig. 3*B*). The changes were particularly striking in the skull, the areas of destruction being very

markedly decreased both in number and extent. Through 1937 and early 1938 she felt entirely well and was able to be up and about. In July, 1938, the patient stated she was losing weight rapidly but continued very active. In September, 1938, the patient committed suicide.

Case vi. R. P., female, aged forty-four, married, two children living and well. In 1939, she had a right radical mastectomy for cancer of the breast. When seen in July, 1940, she showed two recurrent nodules in the scar and stated that these lumps had been present for several months and had been gradually increasing in size. Roentgen therapy was administered with 200 kv. and 0.5 mm. of copper filtration; 3,000 r was delivered over each nodule. Roentgen studies of the chest, skull, pelvis and spine revealed no evidence of metastases. Three months later she returned complaining of backache. Roentgen examination showed metastatic involvement in the neck of the left femur and the inferior portion of the right ilium slightly above the acetabulum. Roentgen sterilization was performed, 1,200 r being administered to each of four pelvic fields, two anterior and two posterior. Also, 1,600 r was delivered to the lumbar area, the left chest posteriorly and the anterior surface of the right shoulder.

When seen in April, 1941, she was entirely free of pain and felt well. In November, 1941, she had a productive cough. Roentgen studies showed the metastatic lesions in the bones filled in. She died in January, 1942. No autopsy was obtained.

Case vii. R. M., female, aged forty-seven, single. Menstrual history uneventful. In June, 1931, she noted a mass about as large as a cherry in the upper portion of the left breast, but did not consult a physician till February, 1933. In June, 1933, she was admitted to the Pondville Hospital with massive involvement of the left breast by scirrhous carcinoma. There was ulceration of the medial aspect of the breast with multiple skin nodules extending in every direction from the area of ulceration. No masses were present in the axilla and there was no edema of the arm. There was fullness in the left supraclavicular fossa. The right breast and axilla were normal. Roentgen studies revealed extensive osteolytic metastases in the skull, the pelvis, and the upper ends of the femurs (Fig. 4*A*), the vertebrae and the shoulder girdles. There were extensive, bilateral pulmonary metastases. Roentgen treatment with 200 kv. and 0.5 mm. copper filtration was administered; 1,200 r to the left breast, 800 r to the pelvis posteriorly and 600 r to the anterior pelvis. During the next two months she remained in bed and appeared to improve slowly with gain in weight and strength. She suffered mild attacks of dizziness with nausea and vomiting, considered to be due to the cranial metastases. In February, 1934, roentgen examination revealed marked regression of the

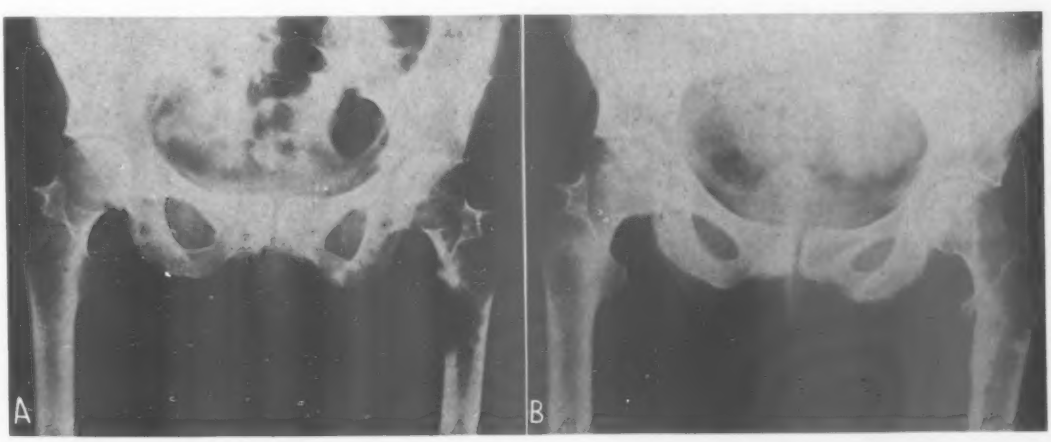

Fig. 4. Case vii. *A*, June 8, 1933. Advanced metastatic carcinoma with multiple areas of destruction in the bones of the pelvis and upper femurs. *B*, December 6, 1934. There has been extensive recalcification of the osteolytic lesions, many of which have disappeared completely. The right ischium and ilium show a return to practically normal contour and bone structure.

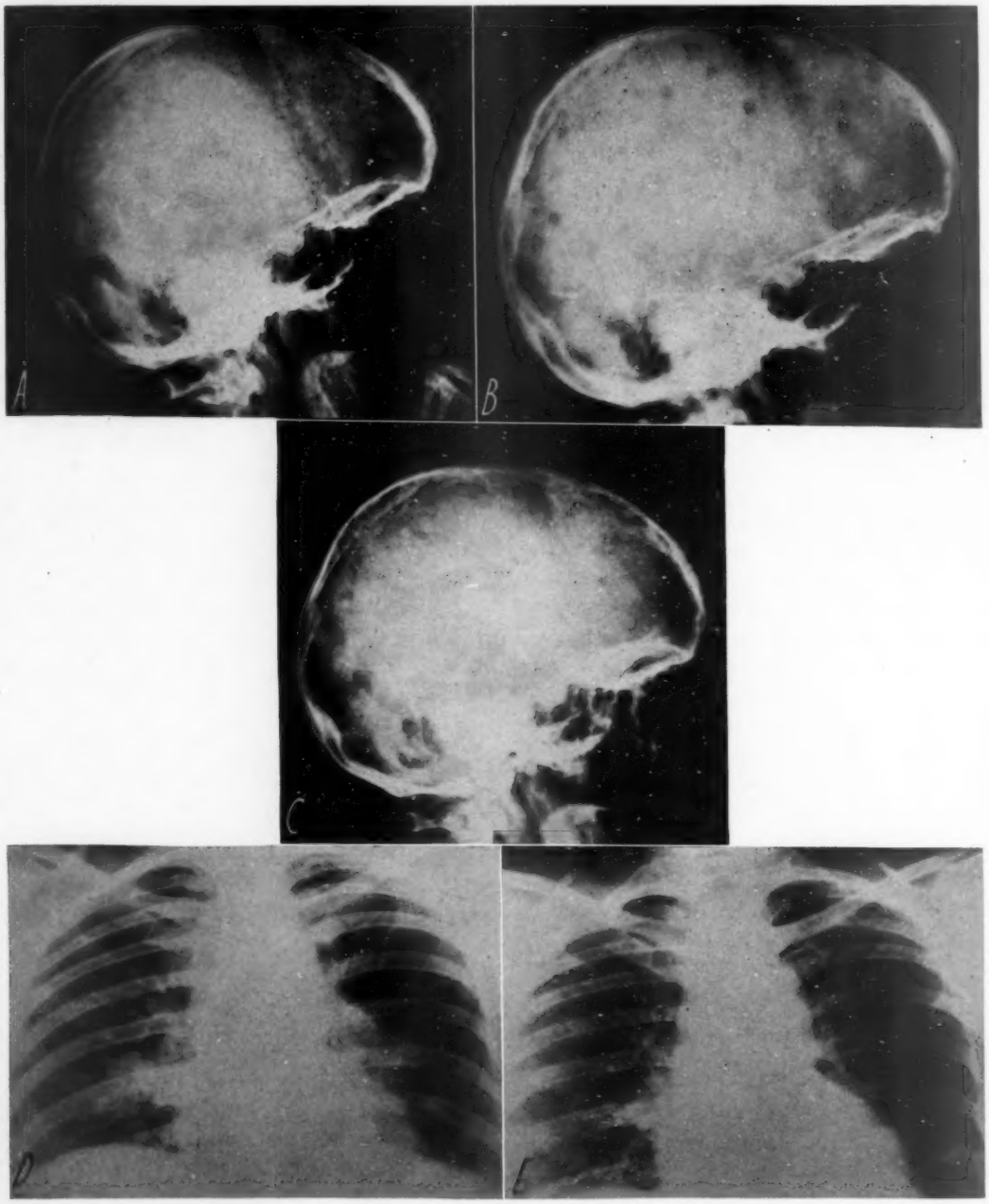

Fig. 5. Case VIII. *A*, February 16, 1933. Several very small foci of metastatic carcinoma are demonstrable in the frontal and parietal regions. *B*, July 20, 1934. There are multiple metastatic lesions scattered throughout the skull. *C*, December 28, 1934. The areas of increased radiance in the skull have decreased markedly in number and size. *D*, February 19, 1933. Advanced metastatic carcinoma involving the right lung, the left base, the mediastinum and the hilar glands. *E*, December 28, 1934. There is now no definite evidence of pulmonary metastases.

bone lesions with extensive regeneration of new bone. Follow-up studies six months later showed further improvement of the bone lesions. In December, 1934, the bone metastases were still well filled in (Fig. 4*B*). Her general physical condition had improved further and she had gained 8 pounds.

In March, 1935, roentgen studies showed re-

currence of the bone metastases. Pain had recurred, especially in the chest and thighs, and she was confined to a wheel chair. Radiation therapy was administered to the left breast, the right shoulder posteriorly and the lumbosacral region. The pain increased and in August, 1935, she suffered a spontaneous fracture of the left femur. The patient died on September 21, 1935. Postmortem studies revealed extensive metastatic disease in the left axilla, chest, liver, adrenals, spleen, ovary and the skeleton.

Case VIII. H. S. A., female, aged forty-six, married. Her menstrual history was uneventful and she was still menstruating when first seen at the Pondville Hospital. In March, 1932, she had a left radical mastectomy because of a slowly growing mass which she stated had first been noticed four years previously. In January, 1933, there was a rapidly growing nodule overlying the upper sternum, and the following month she entered the Pondville Hospital. Roentgen studies revealed bilateral pulmonary metastases (Fig. 5*D*); there were several small areas of increased radiance in the skull (Fig. 5*A*). Roentgen treatment was given, 1,600 r being delivered through a field 15 by 15 cm. over the lesion on the anterior chest wall. The mass disappeared completely within the succeeding few months, but the pulmonary metastases increased and in June, 1933, 12,000 r was delivered over a field 20 by 20 cm. on the lower thorax posteriorly.

In December, 1933, the patient complained of severe pain in the lower back and had a slight cough. Roentgenograms revealed extensive destruction due to metastatic cancer in the bones of the pelvis and the upper femurs. She received 800 r over the lower back with prompt relief. About six months later, there was a recurrence of severe pain in the back and legs. Two small, movable nodules were present in the left axilla. Roentgen studies demonstrated multiple punched-out, osteolytic metastatic lesions in the skull (Fig. 5*B*), and a slight increase in the destructive processes previously noted in the pelvis and femurs. In July, 1934, roentgen therapy was administered as follows: 800 r to the pelvis and lumbar spine and 1,200 r to the left pectoral region. There was considerable relief of pain following the treatments. When seen three months later, she stated that she had had no menstrual periods and was having menopausal symptoms. As there had been a recurrence of pain in the back, a single dose of 400 r was delivered over the pelvis posteriorly.

In December, 1934, she was entirely free of pain. Roentgen studies revealed marked regression of the bone lesions, the skull particularly showing great improvement (Fig. 5*C*). The lungs also appeared free of metastatic disease (Fig. 5*E*). She was observed at frequent intervals and her condition remained satisfactory with some weakness and discomfort in the lumbar region, but no pain. In 1936, a report was received from another hospital that she was suffering from cancer of the rectum, for which a colostomy was performed. Death occurred on June 11, 1938, and the autopsy report was "cancer of the rectum, cancer of the peritoneum, intestinal obstruction, generalized peritonitis, bilateral cysto-ureteral pyelonephritis."

SUMMARY AND CONCLUSIONS

Osseous metastases from cancer of the breast may regress or disappear completely after ovarian sterilization.

The absence or withdrawal of the ovarian hormone is the important factor in producing the results.

In addition to the beneficial effects observed in the bone lesions, there may also occur marked relief of pain, general improvement in the physical and mental status, and the prolongation of the patient's period of usefulness.

The regression of the osseous metastases prevents pathologic fractures with their resultant deformity and disability.

The question as to whether this mode of therapy results in appreciable prolongation of life cannot be definitely answered in the affirmative at present.

Routine roentgen sterilization of all patients with cancer of the breast is not advocated, the treatment being recommended when osseous metastases have been demonstrated by roentgen examinations.

About one-third or more of the patients treated may be expected to show improvement after ovarian sterilization.

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DISCUSSION

DR. C. M. RICHARDS, San Jose, Calif. There is just one other horn of the dilemma that was not brought out in Dr. Ritvo's paper, although he may have mentioned it in his complete paper, and that is that once we have produced a menopause in these patients then we are faced with the dilemma of mitigating the menopausal symptoms. It is our experience that we have to be very watchful where the patient is also under the care of the family physician to see that he does not start giving the patient estrogenic substances to relieve her menopausal symptoms and thus put her back to the place where she is getting her estrogenic substances again, thereby defeating the object that we have obtained in producing the menopause. I think we must be very watchful of that or else our results will be vitiated.

Dr. IRA I. KAPLAN, New York. I am very much interested in this paper because we meet with this question all the time in our work in New York. In the hospital in which I am principally interested, we naturally get the last stage cancer cases and frequently are requested to take care of patients who have extensive skeletal metastatic lesions. We treat them with sterilization whenever we can, but we are not quite sure whether we should or should not stop at the menopausal age because frequently, in spite of the fact that the patient has been through the menopause, if we still irradiate the pelvis over the ovarian areas in a number of instances we do still get recession of skeleton metastasis.

One of the discussants reported a very interesting case in which there was a resolution of the skeletal metastasis but no resolution of the liver metastasis. We find that in numerous cases. While the skeletal metastases respond to sterilization, the visceral metastases do not seem to so respond.

Now there is another important question

and that is the question of cancer of the breast, metastasis of the skeleton, and the presence of pregnancy. What shall we do in that type of case? Shall we interrupt the pregnancy? Shall we sterilize the patient thereby? Shall we interrupt the pregnancy with manual methods and then sterilize the patient or what should we do? That is one of the most important questions that we often encounter in our service.

Dr. Richards mentioned a very important point, which I think should be emphasized constantly, and that is the attempt of the physician to ameliorate menopausal symptoms by estrogenic injections. It just upsets everything you try to do with sterilization for metastatic cancer. It is a rule with us that when we return patients to the physicians or send them back to other clinics we distinctly tell them that estrogenic hormone treatment should not be instituted.

ROENTGEN IRRADIATION IN POLYCYTHEMIA VERA BY MULTIPLE SMALL DOSES TO LARGE AREAS OF THE BODY*

By LAURENCE L. ROBBINS, M.D.
BOSTON, MASSACHUSETTS

ANY METHOD of treatment, particularly one which may be dangerous to the patient, should be checked and evaluated at intervals. It is the purpose of this paper to study a method of roentgen therapy for polycythemia by multiple small doses to large areas of the body, which has been used in the Tumor Clinic of the Massachusetts General Hospital over a ten year period, from 1932 through 1941. This study, confined as closely as possible to the point of view of the radiologist, has resulted in several interesting observations as well as in some modification of the technique of treatment.

In a brief review of the literature the development of roentgen therapy in polycythemia vera may be traced from 1907 when Stengel[5] was the first to advocate irradiation of the bone marrow for this disease. In 1916 Lüdin[5] first successfully irradiated the skeletal long bones of an erythemic patient, and for many years this proved to be the best method of radiation therapy. In 1927 Teschendorf[10] advised irradiation of the entire body for generalized lesions, and in 1932 Sgalitzer,[8] using this method, reported good results in polycythemia. Hunter[3] and Sanderson[7] in 1936 reported 3 cases which are included in the present series. Pierson and Smith[6] believe that this is a satisfactory method of treatment.

Irradiation with multiple small doses given to large areas of the body is the treatment of choice after the diagnosis of polycythemia vera is established. Before treatment, however, there are several points which the radiologist must consider. In the first place, the diagnosis must be definite. It is dependent upon a persistent polycythemia and, in the majority of cases, splenomegaly. Polycythemia due to chronic heart and lung lesions, as well as physiological response to high altitudes, must be eliminated as these should never be treated by irradiation. The percentage of oxygen saturation of the arterial blood is normal in polycythemia vera, whereas it is low in congenital heart disease and in pulmonary lesions. Bone marrow biopsy is of diagnostic value, particularly in the ruling out of leukemic polycythemia, but is seldom necessary.

Table 1 gives an analysis of 20 cases of polycythemia vera treated at the Massachusetts General Hospital during a ten year period. The dosage has been tabulated in round figures and represents the sum of the amounts given to the two fields.

METHOD OF TREATMENT

In the 20 cases under discussion, daily doses of 20 to 50 roentgens were used. Small doses were chosen because, being unlikely to cause roentgen sickness, they might be more valuable than large doses by prolonging the series of treatment. Total doses varied from 200 to 1,200 r per series, the majority of patients receiving 500 to 600 r per series. Because satisfactory remissions were secured from this dosage, it has been continued. The field covers the body from knees to neck. This is accomplished by using a 20 by 20 cm. cone on the machine and a target-skin distance of 215 cm. The field is centered at the level of the crest of the ilium, and alternate anterior and posterior fields are used. The other factors are 200 kv., 0.5 mm. Cu plus 1 mm. Al filter,

* From the Department of Radiology, Massachusetts General Hospital, Boston. Presented at the Forty-third Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 15–18, 1942.

giving a half-value layer of 0.92 mm. Cu. Calculation of the output of the machine is determined with the Victoreen r meter, measured in air, at the actual target-skin distance and by the inverse square law. The present daily dose is 20 to 30 r, for a total of approximately 500 r in one series; that is, 250 r anteriorly and 250 r posteriorly.

Weekly white blood counts are done until a normal range is reached; then they are checked daily. A fall in the white blood cells below 4,000 or 5,000 warrants an interruption of treatment. During the period of treatment the red blood count and hemoglobin do not, as a rule, change greatly, but if at the end of two or three months the red blood count has not fallen satisfactorily the course of treatment may be repeated cautiously.

RESULTS OF TREATMENT

Of the group studied, one patient died early in the treatment (Case 19); one death occurred six weeks after the completion of treatment (Case 6). One patient showed a temporary fall in the red blood count but seven months later a rapid rise occurred (Case 13). One patient has not been followed (Case 4). The remaining 16 patients did very well, showing remissions varying from six months to five years. Two of this group are dead of other causes. It is important to note that those with the longest remissions had a white blood count of 6,000 or less at the completion of treatment.

The patient usually showed no irradiation reaction and began to improve symptomatically while under treatment. Many continued their work during treatment. The white blood count remained low (1,400 to 6,000) for approximately a month after treatment and then gradually returned to normal. The red blood count began to decrease toward the end of the series of treatments and continued for several months at levels of 3.5 to 6.0 million. When the red count rises above 6.5 to 7.0 million the series of treatments should be repeated. The spleen may or may not regress in size, but usually does. The remission of symptoms is variable in length of time, but in our experience is longer on an average than with other methods of treatment.

COMMENT

The dangers of roentgen treatment by multiple small doses to large areas of the body are, as in other methods of irradiation, leukopenia and the possibility of late irradiation anemia. While leukopenia is present the patient may be unable to combat an intercurrent infection, as may have occurred in Case 6. Anemia may develop in untreated cases also and its appearance in treated cases is not necessarily due to treatment. Bleeding duodenal ulcers, for instance, are common in this disease and may cause severe anemia. In the group of cases studied no anemia developed which could be attributed to radiation treatment.

The primary advantage of this method, as compared with other forms of radiation therapy (that is, larger daily doses to the ribs or systematic irradiation of the long bones[4]) is that it gives longer periods of remission with less danger of irradiation damage to tissue. It is no more dangerous; it does not cause roentgen sickness as a rule; and it does not interfere with the patient's daily work. Irradiation of the spleen has no lasting effect either on the symptoms or on the red blood count (Case 1). Irradiation of the pylorus and Brunner's glands has not proved successful.[9] Early reports indicate that treatment with radioactive materials may be of value,[1] but until they have been tested more extensively and until their cost is lower and the supply more plentiful, roentgen treatment with multiple small doses will continue to be of more practical value. Phlebotomy gives good temporary relief, but must be repeated at relatively frequent intervals. Treatment with phenylhydrazine, its derivatives, or arsenic—toxic drugs whose use must be carefully controlled—is of value only while the drugs are taken, and they usually cause the patient marked discomfort.

TABLE I

ANALYSIS OF 20 CASES OF POLYCYTHEMIA VERA TREATED WITH MULTIPLE SMALL DOSES OF ROENTGEN RADIATION AT THE MASSACHUSETTS GENERAL HOSPITAL (1932–1941)

	Case	Sex	Age	Symptoms		Blood Count: Hg	Blood Count: Erythrocytes (million)	Blood Count: Leukocytes	Spleen	Date	Treatment	Oxygen Saturation per cent	Remarks	Length of Remission	Final Status
U-65650	1	M	52	Weakness; headache; bleeding ulcer	B	65% T	5.3	17,300	Enlarged	8/19/32	600 r spleen—each 3 series		No decrease in size of spleen		
					A		6.7	16,500	Enlarged	9/26/32					
					B	100%	10.6	17,400		6/27/33	500 r—30 r daily				
					A		10.3	7,300		7/20/33					
					B	75%	4.9	5,700		9/25/33	1,200 r—50 r daily			1¼ yr.	
					A					10/28/33					
					B		5.7			7/22/35	180 r—30 r daily			2½ yr.	
									Enlarged	2/14/38	600 r spleen				
									Enlarged	8/14/38	600 r spleen				
					B	135% S	7.3	16,600		11/14/38	900 r—30 r daily			2½ yr.	
					A		5.6	4,300		3/—/39					
									Enlarged	9/16/40	600 r spleen				
					B		7.9			6/23/41	500 r—20 r daily				Working; in good health
					A	14.0 gm.	4.7	9,200		12/31/41				6 mo.	
10879	2	F	49	Headache; migrating phlebitis legs and abdomen	B		7.0	12,950	Enlarged	11/25/32	300 r—30 r daily		Red blood count still elevated		
					B		8.1			1/24/33	600 r—20 r daily				
					A	60% T	4.4	4,200	Negative	4/—/33				4 yr.	
					B	100% S	6.2	7,100		4/ 5/37	600 r—25 r daily		Recurrence of phlebitis; improved		
					A		4.5			7/—/37				3½ yr.	
						19.5 gm.	5.8	7,500		7/15/40			Became obese against advice		Sudden death after large meal; coronary; no autopsy
										11/—/40					
129760	3	F	38	Hypertension 300/100; kidney damage	B	100% S	7.3	17,000	Slight enlargement	8/30/32	600 r—20 r daily		Treatment after venesection of 1,300 cc.		
					A		3.7	1,400		10/17/32				5 yr.	
					B	164.0 gm.	6.0	11,300	Slight enlargement	1938	9200 r rt. breast and axilla—7 mo. period		Inoperable cancer (breast)		Died; cancer; cardiac failure
										3/21/40					
H-32338	4	M	54	Paralysis rt. leg and arm; headache; aphonic	B	123% S	7.6	13,900		9/ 6/32	600 r—20 r daily		Venesections—800 cc.; no follow-up. In another hospital Sept., 1933; reason unknown		Unknown
					A		7.2	8,600		10/ 4/32					
30403	5	M	62	Vertigo 8 yr.; cerebral accident 6 mo. before; gangrene of toe on entry	B	103% S	6.6	11,000		4/21/36	400 r—30 r daily		Toe amputated		
					A		5.4	3,100	Negative	6/—/36				3 yr.	
					B		6.0			4/ 7/39	600 r—30 r daily				
					A	14.3 gm.	{4.9 5.5}	6,900		11/ 3/41			Improved and able to work	2¾ yr.	General condition has remained unchanged

(Key—B—Before; D—During; A—After.

	Case	Sex	Age	Symptoms	Blood Count	Hg	Erythrocytes (million)	Leukocytes	Spleen	Date	Treatment	Oxygen Saturation per cent	Remarks	Length of Remission	Final Status
8589	6	M	65	Swelling, discoloration feet and legs	B	130% S	9.9	17,000	Slight enlargement	12/ 8/36	300 r—30 r daily	91.8	Clinical diagnosis, deep thrombophlebitis and cellulitis		
					A		7.5								
					B					2/ 9/37	700 r—50 r daily		After treatment, lumbar puncture; discharged; 4 days later re-entry because of headache. Spiked temp. to 102° F.; frank hemoptysis. Chest *x-ray*-cavities; consolidation rt. upper lobe		Died 6 wk. after re-entry; no autopsy
					A		7.2	3,300		2/25/37					
								1,300		March '37					
								4,000							
40086	7	F	50	Headache; plethora; blood pressure 178/128	B	122% S	8.1	9,500	Negative	5/ 9/37	500 r—30 r daily	95.5	Felt much improved	4½ yr.	Continues to feel well
					A		5.7	6,800							
						16.1 gm.	4.7	10,200		9/ 8/41			Feeling well		
115923	8	M	62	Difficulty walking: unsteady gait						1934	Roentgen treatment to chest for neurofibroma		Pathologic report on biopsy later was hemangioma		
										1938					
					B	130% S	7.2	10,300		5/23/38	500 r—30 r daily	94.9	Improved	3 yr.	
					A		5.4	7,000							
					B		7.9			11/28/38	700 r—30 r daily; discontinued because of leukopenia				
					D			1,800							
					A		6.4	7,100							
					B		7.2			10/27/41	400 r—25 r daily; discontinued (leukopenia)		General condition good		General condition continues good
142886	9	M	55	Gangrene lt. 5th toe; plethora	B	14.9 gm.	6.9	11,000		10/31/38	1000 r—30 r daily	98.0	Toe amputated	3 yr.	Has been well, without further thrombosis
					A		4.8	11,600							
							4.5	12,200		12/7/41					
175527	10	F	68	Known polycythemia with previous hemorrhage after minor operations; lesion on vulva at entry	B	128% S	8.9	21,000	Enlarged	2/16/39	200 r—30 r daily; stopped—hemorrhage from biopsy wound		Had been treated with phenylhydrazine; biopsy (vulvectomy): kraurosis		
					D		4.2			2/23/39					
										4/17/39					
					B	18.3 gm.	7.2	17,400	Slight enlargement	12/ 4/39	500 r—25 r daily			2 yr.	
					A	18.1 gm.	6.9	15,500							
							4.7	8,000		7/—/41			Cerebral accident with paralysis; venesections		Bedridden since July, 1941
163704	11	F	65	Dyspnea; varicose veins; phlebitis; ankle edema; plethora	B	20.8 gm.	7.7	15,600	Enlarged	10/ 9/39	650 r—30 r daily	95.0	Much improved	2 yr.	
					A	19.5 gm.	6.5	5,200							
							4.1			10/—/40					Patient continues to feel well
						15.1 gm.	5.8	12,500		11/29/41					
198686	12	F	61	Dyspnea; weakness; active duodenal ulcer	B	168% S	7.1	5,300		10/23/39	500 r—30 r daily				General condition good
					D			3,800						2 yr.	
					A	20.1 gm.	6.2	6,500							
						16.4 gm.	4.4	6,400		1/13/41					
						17.5 gm.	4.6	6,000		10/20/41					

TABLE I—*Continued*

	Case	Sex	Age	Symptoms	Blood Count				Spleen	Date	Treatment	Oxygen Saturation per cent	Remarks	Length of Remission	Final Status
						Hg	Erythrocytes (million)	Leukocytes							
215738	13	M	47	Hemoptysis 15 yr.; plethora	B	132%	8.5	20,400	Enlarged	11/ 6/39	250 r—30 r daily	91.0 95.0	Sternal biopsy unsatisfactroy	None	
					A	21.8 gm.	9.2	12,700		12/ 4/39 12/21/39	350 r—25 r daily			None	
					B A	16.9 gm.	8.6 5.8	8,400		2/ 6/40 6/—/40	400 r—50 r daily; not completed		Lack of cooperation		
						24.3 gm.	8.1			9/—/40			Venesection—500 cc.		
							8.5			1941			Same condition. Refused further treatment of any sort		Spring, 1942 returned for treatment
226399	14	M	64	Gangrene rt. foot and leg; mental confusion and aphasia 5 mo.	B A	128% S 12.7 gm.	7.7 4.4	14,400 6,100	Enlarged	12/18/39	500 r—25 r daily		Leg amputated; mental condition cleared	2 yr.	
							5.0 5.0			1940 Dec. 1941					Remained relatively normal; feeling well
253145	15	M	53	Peptic ulcer for years; coronary thrombosis 9 mo. before entry; plethora	B A	17.5 gm.	8.3 6.0 5.2	18,500	Enlarged	7/22/40 1/—/41 10/—/41	250 r—30 r daily; not completed; (became blood donor)		Anginal pain persists		Angina persists; no longer acting as blood donor
270452	16	F	37	Known polycythemia over 6 yr.						1934 to 1940	Phenylhydrazine and localized irradiation to spleen and long bones		Nausea and vomiting from drug. Treatment caused temporary falls in red count		
				Migraine attacks	B A	21.4 gm.	7.5 3.3	10,000 4,400	Enlarged Smaller	12/ 9/40	500 r— 25r daily		Some dizziness; generally better	1 yr.	
						20.7 gm.	6.0	5,700		12/22/41					Still some vertigo; improved
287060	17	F	57	Vertigo; blurred vision; paresthesia extremities; nausea; vomiting	B A	22.5 gm.	7.4 4.9	10,000 5,100		2/13/41	500 r—25 r daily	95.0	Symptoms of 2 yr. duration Lowest leukocyte count noted 4,300		
						14.9 gm.	5.1	7,200		11/17/41				8 mo.	In good condition
206930	18	M	40	Headache 3 yr.; lethargy; anorexia; peptic ulcer	B A	17.5 gm. 14.5 gm.	10.5 4.9	12,000 4,600	Enlarged	6/23/41 9/—/41	500 r—20 r daily	89.0		5 mo.	Has remained well
324084	19	F	74	Gangrene tip rt. index finger	B	90%	10.5	22,000		10/20/41	150 r—30 r daily		Had had several venesections (unknown amounts)		Was discharged to home. Nurse called to bring her to hospital; found her dead. Cause unknown
126422	20	M	76	Headaches; vertigo; diabetes	B A	24.0 gm. 11.0 gm.	9.0 4.7	17,000 6,000		6/ 9/41	320 r—20 r daily		Improved in general health	6 mo.	Remains in good condition

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CONCLUSIONS

A study of 20 cases of polycythemia vera treated at the Massachusetts General Hospital during the past ten years with small doses of roentgen radiation to large areas of the body has emphasized two important advantages of this method of treatment: first, it caused no definite ill effects; second, it resulted in longer remissions than had followed other forms of therapy. The present technique of treatment is the most satisfactory which has been developed.

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Representatives on American Board of Radiology: Douglas Quick, New York, N. Y., B. P. Widmann, Philadelphia, Pa., F. W. O'Brien, Boston, Mass.

Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIALS

PERFORATED PEPTIC ULCER

IN 1942 there was editorial comment[1] in this JOURNAL on the "Effect of heavy air raids on the incidence of perforated peptic ulcer." This editorial was based on a report of Stewart and Winser. In order to confirm their impressions that there was a definite rise in the incidence of perforated peptic ulcer during air raids, they[2] have again analyzed statistics dealing with this problem from the same group of hospitals and they have come to the conclusion that the increase in perforated peptic ulcer during the period of heavy raiding was statistically significant when compared with the previous three, and the following two years. They have statistical evidence which seems to prove or certainly to favor some general tendency having operated during the air raid period to cause perforations of peptic ulcer and definitely in the two years following the general blitz period there has been a steady decline in the number of perforated peptic ulcers entering the hospitals.

Recently Raw[3] has analyzed a series of 312 cases of perforated gastric and duodenal ulcer and in a discussion of his cases he points out the fact that perforated ulcer should be regarded as one of the most important of abdominal diseases, and that in the particular area in which his statistics were gathered the total number of deaths is considerably greater than the total for appendicitis, though the latter condition is more common. As might be expected, the patients are usually in the prime of life and the fatality rate varies greatly with early diagnosis and the experience of the surgeon in handling such cases. Raw describes the clinical picture which such cases present at the time of perforation and also calls attention to the aid which the roentgen ray can render in such cases. As has been pointed out by a number of observers, the fatality rate runs closely parallel to the number of hours between perforation and operation, and the mortality rate increases with the more advanced age of the individual. While perforations in women are fairly uncommon in his group of cases, as in other statistical studies, it is in women that the diagnosis is so often not made early because in many of these cases there is a mistaken diagnosis of gallstones and the patient's operation is delayed and consequently the probability of a cure from operative procedures is lessened.

In Raw's series of 312 perforated gastric and duodenal ulcers there was a gross mortality of 14.4 per cent and the two most important factors influencing this operative mortality were the age of the patient and the interval before operation.

Since the operation for perforated peptic ulcer is fraught with considerable initial mortality rate and a rather high morbidity rate, it is important to know some of the late results of perforated peptic ulcer. Since the subject has assumed such great importance, particularly in the zones of combat and the cities where bombing has been carried on to a certain great extent, it is of interest to learn the late end-results in a group of cases covering a period of ten years. Such a group of cases was analyzed by Wakeley[4] at the Royal Naval Hospital, Haslar, and a ten year period was chosen

[1] Editorial. Effect of heavy air raids on the incidence of perforated peptic ulcer. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *47*, 626–627.

[2] Spicer, C. C., Stewart, D. N., and Winser, D. M. de R. Perforated peptic ulcer during the period of heavy air-raids. *Lancet*, Jan. 1, 1944, *1*, 14–15.

[3] Raw, S. C. Perforation of gastric and duodenal ulcers. *Lancet*, Jan. 1, 1944, *1*, 12–14.

[4] Wakeley, C. P. G. Late results of perforated peptic ulcer. *Lancet*, Jan. 1, 1944, *1*, 11–12.

from 1924 to 1934. There were 103 cases admitted to the hospital during this time and all were submitted to operation, with a case fatality of 8 per cent. This is a remarkable figure in contrast with some of the higher mortality rates reported from general civilian hospitals. Part of this low mortality rate is due no doubt to the fact that the average age of the patients was considerably lower than that encountered in civilian practice. The average age of the patients at the Royal Naval Hospital was thirty-two years and, as might be supposed, the men were in a fairly fit condition apart from the fact that they had peptic ulcer. Wakeley attributes another factor to the lower mortality rate, namely the short interval between the perforation of the peptic ulcer and operative interference, the average time being four hours.

In contrast to this group of cases that were cared for in the Royal Naval Hospital, Wakeley collected 102 cases of his own, both hospital and private patients, over the years 1924 to 1934. The average age in this group was forty-six years and the operative mortality rate was 20 per cent. This increase in the mortality rate in the latter group of cases he attributes to the poor condition of the patients themselves and the long period which elapsed between the actual perforation of the ulcer and the time of operation, an average of ten hours.

In the follow up of the group of cases which were operated upon at the Royal Naval Hospital only 5 per cent of the group of cases were untraced. It is of interest in passing that in the Service cases 54 per cent and in the civilian cases 51 per cent of the perforations were gastric in origin.

One interesting and important feature of Wakeley's investigation of the Royal Naval Hospital cases was the fact that 44 per cent of the perforated peptic ulcer cases are still serving in the Royal Navy, the longest time since operation being nearly twenty years and the shortest time nine years. He deduces from a study of his cases in the Royal Naval Hospital that the perforated peptic ulcer patient should not be invalided from the Service but given a careful convalescence, as he is likely to continue to be a useful member of the Royal Navy.

Wakeley also points out the fact that the prognosis is better in perforated cases than in any other form of peptic ulcer. In his group of patients the recurrence of pain or other symptoms was unusual and the patients have lived an ordinary life.

From the analysis of his cases Wakeley concludes that the tendency to reject all men who have had a perforated peptic ulcer is quite unwarranted considering that a high proportion have never had a sign or symptom of their former trouble since operation. In the Royal Navy today, he says, it is quite uncommon to lose a case of perforated peptic ulcer and the operative mortality is about 1 per cent.

Another important feature in his group of cases is that the incidence of subphrenic abscess following perforated peptic ulcer is reduced to the vanishing point.

It may readily be seen that given a group of cases where a correct diagnosis is made and the proper handling of the patient is then carried out with a high degree of efficiency, the mortality rate is greatly reduced. It only remains for an improvement to be made in the diagnostic side of the problem. Certainly the roentgen ray can, when properly employed, play an important part in the diagnosis of perforated peptic ulcer and those not familiar with the various methods of examination by this means should acquaint themselves with a working knowledge of all the various suggestions which have been advanced for the proper examination of the patient with suspected perforated peptic ulcer.

CUMULATIVE INDEX OF *RADIOLOGY*

THE Radiological Society of North America has just issued a Cumulative Index of their official publication, *Radiology*, covering the years 1923 to 1942, Volumes 1 to 39. In the foreword Doub says that "for a number of years there has been a laudable desire to publish a cumulative index of all the material contained in *Radiology* so that it might be more readily available for reference." The final publication of this Index now in reality makes available the enormous amount of literature which has appeared in the thirty-nine volumes of *Radiology* on radiological and allied subjects. This Cumulative Index fills a great need because *Radiology* has always occupied a position in the forefront of radiological publications and it will prove of immense value to radiologists and medical men the world over. In the organization of the Index the editorial staff has followed the same arrangement of subjects and authors as the *Quarterly Cumulative Index Medicus*. The Index is attractive in its general appearance, style of type and printing.

The publication of the Cumulative Index of *Radiology* is a happy conclusion of a plan which was formulated several years ago. The editorial staff and the collaborators in the work of publishing this Index are to be congratulated on the completion of an arduous task in such a fine manner.

Apex Newsphotos, Fort Worth

L. W. BAIRD
1907-1943

CAPTAIN L. W. BAIRD died of coronary sclerosis on October 6, 1943, at the Station Hospital in Camp Carson, Colorado Springs, Colorado. He was born April 19, 1907, in Edwardsville, Illinois, the son of Henry N. Baird and Maude White Baird. He graduated from the Edwardsville High School, and obtained his M.D. degree from the University of Illinois in 1932. He spent one year as intern at the Santa Clara County Hospital, also one year as resident at this hospital in San Jose, California. He had a three year Fellowship in Radiology at the Mayo Clinic, and obtained a degree of Master of Science in Radiology from the Graduate School of Minnesota. He left the

Mayo Clinic in December, 1936, and was radiologist at the Scott & White Clinic and the Santa Fe Hospital in Temple, Texas, from then until he entered the Army as a Captain in the Medical Corps in November, 1942. He was stationed at Camp Carson as head of the Department of Radiology from then until the time of his death.

Dr. Baird was a member of the Bell County Medical Society, the Texas State Medical Society, and the Radiological Society of North America. He was a Diplomate of the American Board of Radiology, and a member of the American College of Radiology. He also served as Secretary of the Texas Radiological Society from 1941 to 1942. He was a member of the Phi Beta Pi Medical Fraternity and belonged to the First Presbyterian Church in Temple, Texas. He was married to Helen Keshner on March 31, 1934 in San Jose, California. He is survived by his wife and one son, Robert White Baird. His mother, Mrs. H. N. Baird of Edwardsville, Illinois, and a sister, Mrs. Margarete Whiteside of Fort Worth, Texas, also survive.

Dr. Baird had written scientific papers and appeared on local, state and national programs. He conducted a refresher course on "Carcinoma of the Colon" for members of the Radiological Society of North America at their annual meeting in California in December, 1941.

C. A. Stevenson

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: To be announced.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: 1944, to be announced.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: To be announced.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave., Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Fine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. P. C. Schnoebelen, 462 N. Taylor Ave. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Herman Klapproth, Sherman, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October throughMay, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

NEW OFFICERS

The following officers of the American Roentgen Ray Society were recently elected for the year 1943–1944. The vote of the Society's members was taken by mail since the annual meeting had been postponed because of the World War. *President:* Dr. Sherwood Moore, St. Louis, Missouri; *President-Elect:* Dr. Lyell C. Kinney, San Diego, California; *First Vice-President:* Dr. Paul C. Hodges, Chicago, Illinois; *Second Vice-President:* Dr. Aubrey O. Hampton, Boston, Massachusetts; *Secretary:* Dr. H. Dabney Kerr, Iowa City, Iowa; *Treasurer:* Dr. J. Bennett Edwards, Leonia, New Jersey; *Historian:* Dr. Ramsey Spillman, New York, New York.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION

At the meeting of the Radiological Section of the Los Angeles County Medical Association, held on December 8, 1943, the following officers were elected for the ensuing year: Dr. Benjamin H. Sherman, *President;* Dr. Donald R. Laing, *Vice-President;* Dr. Roy W. Johnson, *Secretary;* Dr. Henry Snure, *Treasurer.*

SAN DIEGO ROENTGEN SOCIETY

Through the efforts of Dr. Keener, Senior Medical Officer in Roentgenology at the Naval Hospital, San Diego, California, a group of radiologists met during the latter part of 1943 and organized the San Diego Roentgen Society to include the local radiologists and those who are stationed in the vicinity in the various Services. The group will meet on the first Wednesday of each month for dinner to be followed by a case seminar. Dr. Henry L. Jaffe of Chicago was elected Secretary. Dr. Jaffe is stationed at the Naval Hospital, Balboa Park, San Diego.

DIRECTORY OF MEDICAL SPECIALISTS

Announcement is made that the Directory of Medical Specialists is now to be published by the A. N. Marquis Company of Chicago, publishers of "Who's Who in America." Previous editions have been published for the Advisory Board for Medical Specialties by the Columbia University Press of New York City.

It is planned not to issue the next edition before 1945, on account of the war, but the A. N. Marquis Company will publish a supplemental list of all those who have been certified by the American Boards since the last (1942) edition of the Directory, totaling about 3,600. This is to be distributed at cost, and monthly or bimonthly bulletins listing successful candidates for certification at examinations during the additional interim before the next edition are to be issued as a subscribers' service.

Dr. Paul Titus (Pittsburgh) of the American Board of Obstetrics and Gynecology will continue as the Directing Editor, and Dr. J. Stewart Rodman (Philadelphia) of the American Board of Surgery continues as Associate Editor. The Editorial Board will be composed, as before, of the Secretaries of the fifteen American Boards.

Communications should be addressed to the Directing Editor, Directory of Medical Specialists, 919 No. Michigan Avenue, Chicago (11), Illinois.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Occupational Tumors and Allied Diseases. By W. C. Hueper, M. D., Assistant Director and Principal Pathologist, Warner Institute for Therapeutic Research, New York City. Cloth. Price, $8.00. Pp. 896. Springfield, Illinois; Charles C Thomas, 1942.

The author has set down in 860 pages an exhaustive review of the world's literature pertaining to the occupational etiology of tumors of the human body. Neoplasms of the various organs and anatomical systems are considered in order and the scope of the book is indicated by a subject index of 46 pages and many elaborate bibliographies, each containing hundreds of references. In one of the final chapters the relation of occupational neoplasia to the various theories of cancerigenesis is considered at some length.

With so little actually known about the cause of cancer the reviewer finds it difficult to justify the labor involved in the preparation of this massive treatise but the author explains that it is intended not only for physicians but for governmental agencies, legislative bodies, workmen's compensation boards, lawyers, engineers, life insurance executives, biologists, and chemists and for this reason many detailed technical data are included. Occupational hazards are stressed throughout the book and the concluding chapter deals with the medicolegal and public health aspects of the subject.

Charles L. Martin

Clinical Roentgenology of the Cardiovascular System. By Hugo Roesler, M.D., F.A.C.P., Associate Professor of Roentgenology and Cardiologist, Department of Medicine, Temple University School of Medicine; Cardiologist, Temple University Hospital, Philadelphia. Second Edition. Cloth. Price, $7.50. Pp. 480, with 337 illustrations. Springfield, Illinois: Charles C Thomas, 1943.

The second edition of this authoritative book is even more comprehensive than the first. Adequate abstracts of the newer diagnostic procedures are given for arteriography, venography, Robb-Steinberg opacification of the cardiac chambers and great vessels, cinematography, kymography and for the detection of intracardiac deposits of calcium. The chapter on Cardiac Measurement includes a thorough discussion of various complicated prediction formulae that will be of interest to those familiar with higher mathematics. New and interesting studies are quoted on the influence of athletics, of avitaminosis, and of the metabolic diseases on the heart. The chapters on Cardiovascular Disease from the Viewpoint of Structural Changes, on Diseases of the Aorta, and on Pericarditis are full of practical clinical observations. The use of small numbers to identify the features of the new illustrations makes them much easier to read and is a distinct advantage.

In general the volume has been brought up to date with emphasis rightly placed on the newer roentgenographic procedures in the study and diagnosis of cardiovascular disease. This progressive viewpoint together with the greater use of the contributions of medical men of this country with more references to our accessible literature is a decided improvement and makes the second edition a most worth-while text. It should be read by all those interested in heart and vascular diseases.

Wendell G. Scott

A Manual of Radiotherapy. By Murray M. Friedman, M. D., Assistant Professor of Radiology, College of Physicians and Surgeons, Columbia University; Assistant Radiologist, Presbyterian Hospital, New York. Paper. Price, $1.50. Pp. 86, with 30 illustrations. Ann Arbor, Michigan: Edwards Brothers, Inc., 1942.

This small paper bound manual gives in brief outline form the factors and dosages used in the Department of Radiotherapy at the Presbyterian Hospital in New York. A short description of each lesion is followed by details of the technique recommended and a limited bibliography. The radium dosage tables and the curves of Paterson and Parker and of Quimby are included, and a few pages are devoted to roentgen injuries and irradiation sickness.

The student will find this outline a valuable guide, but its brevity constitutes its main defect. Certainly the beginner should receive added information before undertaking the treatment of patients. Some of the roentgen irradiation techniques lack the boldness needed for the best results, and such important malignant conditions as cancer of the pharynx, cancer of the cheek and large cancers of the skin are not considered at all. The book could also be improved by the addition of a chapter on the physics of roentgen-ray dosage and more detailed information relative to the practical application of the curves and tables covering radium dosage.

Charles L. Martin

Vol. 51, No. 2

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave. Charleston, S. C.

A SIMPLE METHOD FOR THE ACCURATE MEASUREMENT OF INTRATHORACIC SHADOWS

By G. S. PESQUERA, M.D., *and* E. C. LASHER, R.T.
Metropolitan Life Insurance Company Sanatorium, Department of Roentgenology
MOUNT McGREGOR, NEW YORK

THE accurate determination of the location and the size of objects observed as shadows in roentgenograms of the chest has been attempted by many investigators in the field of roentgenology. The structures that have received particular attention have been foreign bodies, cavities, tumors, and the heart. Their approximate size and location have been derived from the use of various roentgenological techniques together with the aid of triangulation and computed formulae. The methods most frequently used for heart measurements have been the transverse diameter of heart shadows from roentgenograms of the chest taken at 6 or 7 feet (target-film distance), from the tracings obtained from the orthodiagram, and of late by the kymograph.

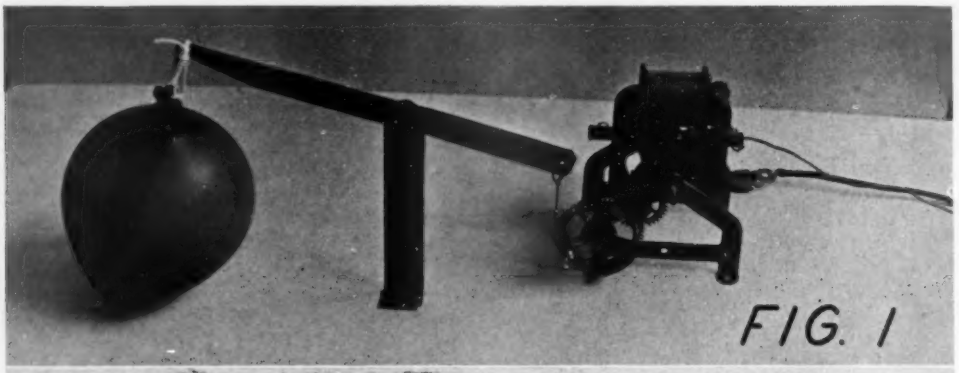
Body section roentgenography is accomplished by making a series of roentgenograms of a subject while the roentgen tube and the film are moved about a fulcrum placed at the level of the section being roentgenographed, the fulcrum being varied for each section taken and varied as many times as there are sections desired. This motion causes gross blurring of all structures except those within the section at the level of the fulcrum. This procedure makes it possible for the roentgenologist to determine with accuracy the distance between the film and the object under consideration. The body section roentgenogram which shows the sharpest image of the object is a roentgenogram of a thin section parallel to and at the same distance from the table top as is the object. Once this factor is determined, the object-film distance is obtained by the addition of the table top to film distance. The difference between the known target-film and the derived object-film distance gives the target-object distance.

Andrew and Warren have evolved a simple formula for the computation of the actual size of any object being roentgenographed when the target-film distance, the target-object distance and the image size are known:

$$\frac{\text{object-size}}{\text{image-size}} = \frac{\text{target-object distance}}{\text{target-film distance}}.$$

We present herewith a simple, and, we believe, an accurate method of measuring objects such as intrapulmonary cavities and foreign bodies, and suggest it as a method for determining heart size. If this method provides a correct answer for object size, then a permanent record obtained at one date may be confidently compared with a record of the same object obtained at any future date. Since the object itself is measured, then such variable factors as gain or loss in weight or changes in posture would no longer need to be considered in the calculation.

We have undertaken to determine the accuracy of measurements with the above method by placing coins of various sizes on blocks at different levels above the roentgen-ray table top. A series of body



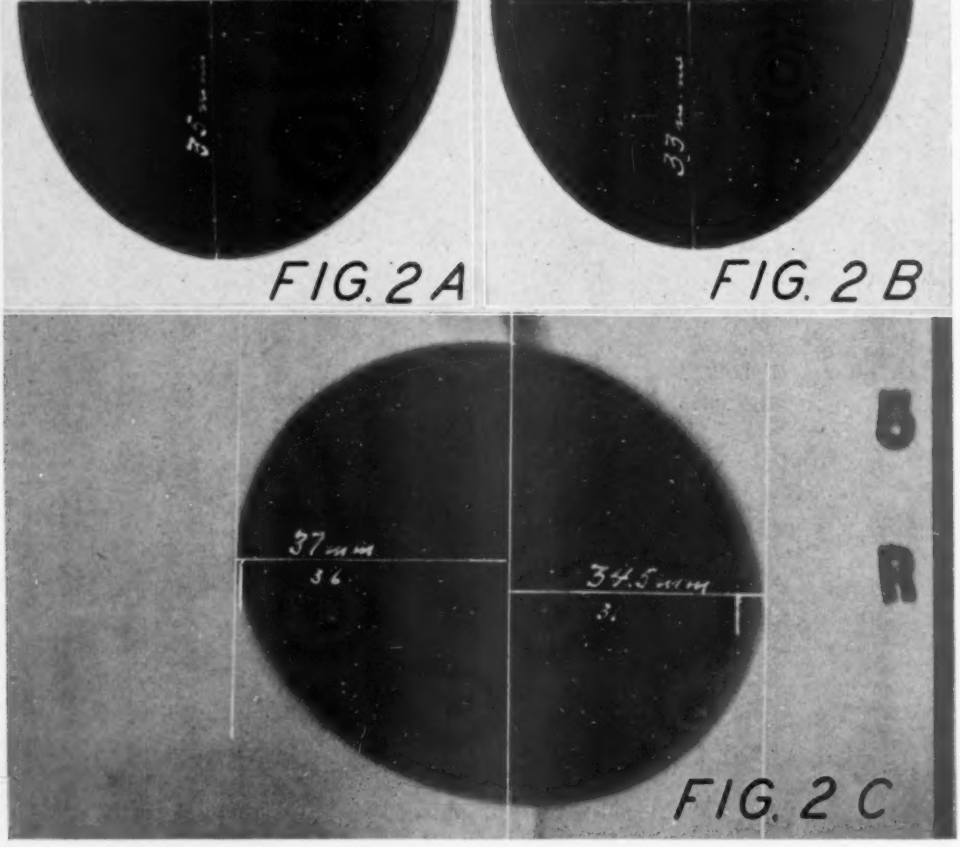


FIG. 1. Model of artificial or simulated heart.

FIG. 2. Body section roentgenograms of heart showing standard cardiac transverse diameter measurement. *A*, diastole, stationary; *B*, systole, stationary; *C*, diastole and systole, heart in motion.

section roentgenograms were then made and from these the one presenting the sharpest image of the coin was selected.

One example will be sufficient to illustrate the technique employed. A five-cent piece measured by micrometer was found to be 2.12 centimeters in diameter. The roentgenogram which gave the sharpest shadow of this coin was taken at 13.5 cm. from the table top. Adding the distance from the table top to the film, usually called Bucky distance when a Potter-Bucky diaphragm is used, we have an object-film distance of 18.5 cm., which when subtracted from the target-film distance of 101.6 cm. gives a target-object distance of 83.1 cm. The image size of the coin measured 2.6 cm. in diameter. Applying the formula of Andrew and Warren we have:

$$\frac{x}{2.6}=\frac{83.1}{101.6}$$

where x equals the object size.

The size of the coin as obtained from this equation was 2.126 cm. which gives an error of 0.006 cm. as compared with the actual measured size of 2.12 cm. The maximal error we encountered in many such calculations was 0.035 cm. We believe that the errors are due to our inability to measure, by the instruments employed, the shadow on the roentgenogram with an accuracy greater than ± 0.025 cm.

To simulate a heart in motion a model was made (Fig. 1) consisting of a rubber balloon filled with an opaque solution (potassium iodide) attached to a fixed base and to a movable arm activated by a small electric motor. With the arm in motion, the balloon was alternately stretched and relaxed at the rate of 70 times per minute. When fully stretched the balloon measured 6 cm. in greatest diameter, and when fully relaxed, 6.4 cm. Body section roentgenograms were made with the object stationary at its smallest diameter (systole), and at its largest diameter (diastole), as well as with the simulated heart in motion. The roentgenograms selected for measurement were those showing the largest and sharpest shadows, since we desired the maximum diameter of the object.

For diastole the roentgenogram selected (Fig. 2*A*) was taken at the 4.35 cm. level from the table top. Adding the Bucky distance of 5 cm. we have an object-film distance of 9.35 cm. When this is subtracted from the target-film distance of 101.6 cm. a target-object distance of 92.25 cm. is obtained. The image (heart silhouette) was measured by drawing a central vertical line from which horizontal lines were drawn at right angles to the outermost point of the curve of each semicircle. The measurement obtained was 7.1 cm.

$$\frac{x}{7.1}=\frac{92.25}{101.6}$$

From the formula the size obtained was 6.446 cm. whereas the actual size was 6.4 cm.

The roentgenogram selected for systole (Fig. 2*B*) gave an object-film distance of 9.9 cm. which left a target-object distance of 91.7 cm. The image size was 6.7 cm.

$$\frac{x}{6.7}=\frac{91.7}{101.6}$$

The calculated size was found to be 6.047 cm. and the measured size was 6.0 cm.

Body section roentgenograms taken with the model in motion (Fig. 2*C*) gave approximately the same results: Diastole, 6.446 cm.; systole, 6.0405 cm.

SUMMARY

Evidence is submitted to show that the actual size of an object is determinable within narrow limits when body section roentgenography and the simple formula devised by Andrew and Warren are employed. For practical purposes the discrepancy between computed and actual size may be ignored. We have also shown that for practical purposes the measurements of a simulated heart in motion are as accurate as when the object is stationary. We suggest the use of this method when accurate transverse measurements of the heart are desired.

We wish to express our gratitude to Dr. E. M. Medlar of our Research Laboratory for his constructive criticism.

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AN EASY METHOD OF FOREIGN BODY LOCALIZATION READ DIRECTLY FROM THE FLUORSCOPIC SCREEN*

By ERNEST LANDY, M.D.
Veterans Administration
SALT LAKE CITY, UTAH

THE following is an easy method by which the depth of a foreign body can be read directly during the roentgenoscopic procedure from a scale placed upon the screen.

The mathematical principles of the method are as follows:

This is illustrated in Figure 1. The principle involved is to obtain a certain angle of rays from the focal spot of the tube with a specified distance between two objects, e.g., the skin marker and the foreign body. This can be obtained as follows: In the line of the central ray of the focal spot, place a metallic marker, *M*, over the position of the foreign body, *FB*. Opposite the marker, *M*, place another marker, *M'*, at a certain distance, e.g., $\frac{1}{4}$ inch, on the same horizontal plane. Now shift the tube and screen so that the shadow of the foreign body, *FB*, will be superimposed on the shadow of the second marker, *M'*, on the screen. The distance of this shift, *OB*, from the central line of rays on the screen establishes a relationship between the depth of the foreign body, *FB*, from the markers. The depth of the foreign body, *FB*, from the markers determines the angle at which the rays from the focal spot with which the shadow of *FB* will be superimposed on the shadow *M'*. This can readily be seen from Figure 1. This angle will always be the same no matter what the position of the foreign body, *FB*, and markers, *M* and *M'*, are in relationship to the focal spot and screen, providing the distances between *FBM* and *M'* and *M* are kept constant. This is shown by the broken lines in Figure 1, when the foreign body *FB* and the markers are placed in a different position in reference to the tube and screen and all the other factors are kept constant.

Triangle *X* will be equal to triangle *Y* by construction. From Figure 1 it will also be seen that triangle *X* will be similar to triangle *OBFS* in the broken lines and it will also be similar to triangle *O'B'FS*, as the angles formed by the parallel lines will

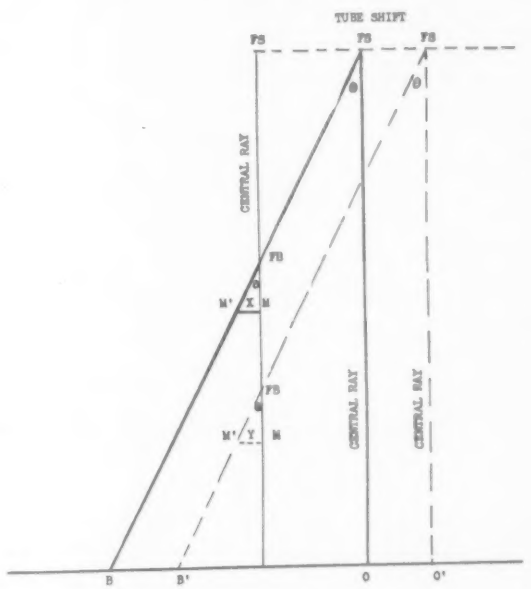

FIG. 1. The markers *M* and *M'* are placed at a certain distance from each other on the same horizontal plane, and at a certain distance from the foreign body *FB*. The figure illustrates that a certain angle θ will be obtained with each distance between the foreign body and *M* and *M'* forming equal and similar triangles. The figures in the broken lines illustrate that if the distance between *FB* and *M* and *M'* is placed in a different position in reference to the focal spot but the distance between them is kept constant, the distance *OB* and *O'B'* will be equal although the tube shift is different.

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be equal. The right angle triangles, *OBFS* and *O'B'FS*, will be equal, as is evident from the figure, as *FSO* and *FSO'*, the focal screen distances are the same and parallel. The lines, *FSB* and *FSB'*, are parallel and equal, and therefore the angles θ will be equal. The distances, *OB* and *O'B'*, will be equal. The distance of the shift on the

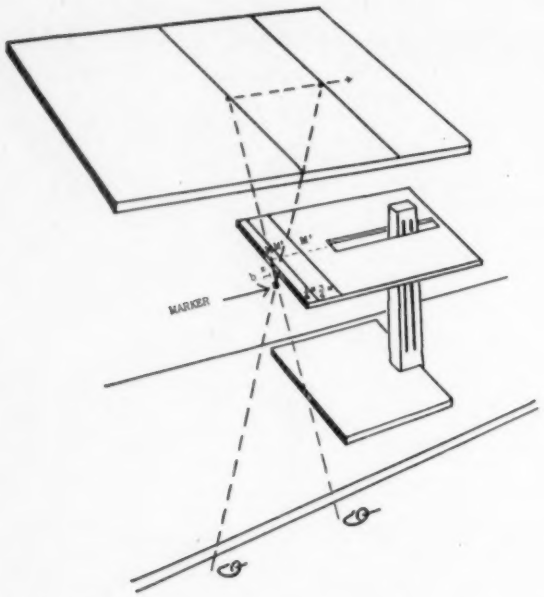

FIG. 2. The apparatus shows how the cross-board with its three wires $\frac{1}{4}$ inch, $\frac{3}{4}$ inch apart with the 1 inch projecting piece (*b*) can be raised or lowered or moved in a horizontal position over the patient. The wires are used as guides so that their shadows will project on the screen upon which a scale can be placed and read. The end wire is centered with the central line of the screen and with the foreign body and the tube and screen shifted so that the shadow of the foreign body projects on one of the wires.

screen *OB* is directly proportional to the angle θ, which is proportional to the distance *MFB*. Each distance between *FB* and *M* and *M'* will give a certain definite angle θ and hence a relationship is established between the depth of the foreign body and the shift at which the marker *M'* will be superimposed on the screen. It is also seen from Figure 1 that although the distance of the tube shift will be different if the constant depth of the foreign body and markers are varied in the relationship to the focal spot and screen, the shift at which the shadow of *FB* and *M'* will be superimposed will be the same on the screen. It is a simple matter to calculate the shift *OB* for any depth between the markers and the foreign body.

It follows, since the triangles are similar, that *FBM* the depth of the foreign body: *FSO* the tube screen distance = *MM'*, the distance between the markers: *OB* the shift at which the shadow of *M'* and *FB* superimpose on the screen. Taking various depths between the foreign body and marker and substituting them in the above equation and selecting a focal spot screen distance, the shift *OB* can be calculated mathematically.

PRACTICAL APPLICATION OF THIS PRINCIPLE

A simple apparatus that can be constructed without difficulty by any carpenter, cabinet maker or machinist is used. This consists of a stand (Fig. 2) about 9 inches high and an adjustable wooden

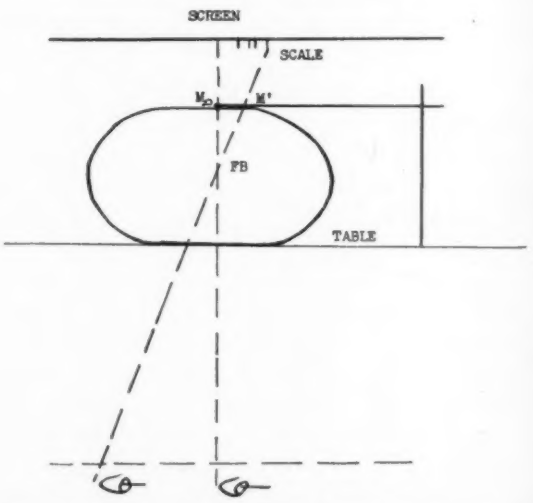

FIG. 3. To illustrate the method and procedure, the cross-bar is lowered so the projecting piece (*b*) is directly over the foreign body. The shadow of the end wire, the center line of the screen and the foreign body are superimposed and the tube and screen shifted until the shadow of the foreign body is superimposed on that of one of the guide wires and the depth read directly from a scale placed on the screen by the reflected light.

cross-bar about 8 or 9 inches wide that can be raised or lowered or moved in a horizontal direction. Upon the cross bar are placed three wires, $\frac{1}{4}$ inch and $\frac{3}{4}$ inch apart. Over the center of the first wire is placed, on the under surface, a projecting piece (*b*) 1 inch from the top of the first wire. During the localization procedure the cross-bar until the projecting piece (*b*) is directly over and upon the shadow of the needle. Then center the center line of the screen with the shadow of the end wire of the cross-bar and the shadow of the needle, so that these three shadows superimpose (Fig. 2). Now shift the tube and screen so that the shadow of the needle is super-

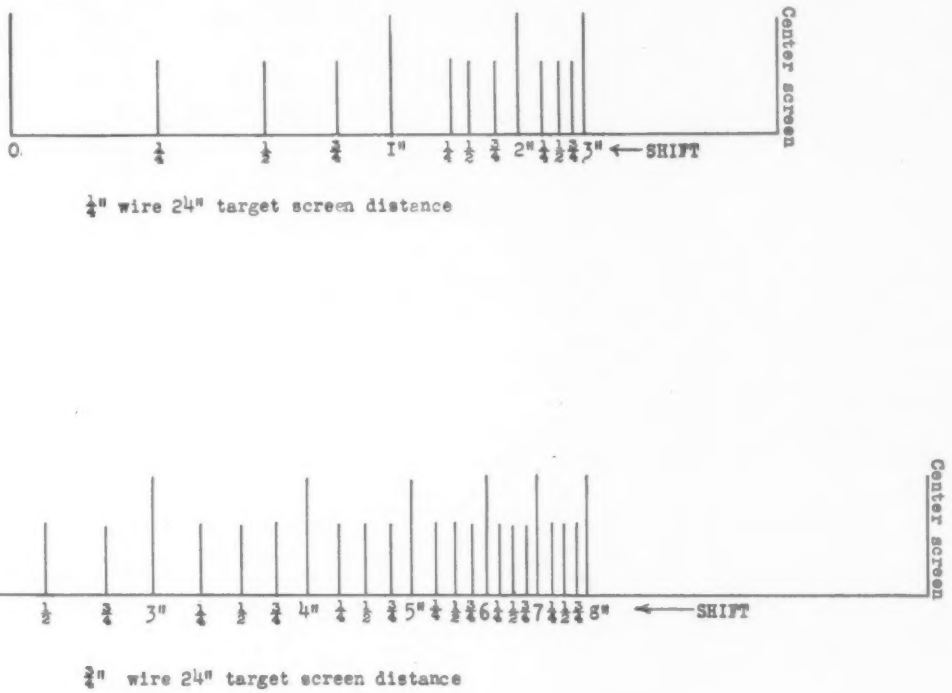
Center screen
0
¼
½
¾
1"
¼ ½ ¾ 2" ¼ ½ ¾ 3" ← SHIFT
¼" wire 24" target screen distance
Center screen
0 ¼ ½ ¾ 3" ¼ ½ ¾ 4" ¼ ½ ¾ 5" ¼ ½ ¾ 6 ¼ ½ ¾ 7 ¼ ½ ¾ 8" ← SHIFT
¾" wire 24" target screen distance

FIG. 4. Two scales are shown, one being the $\frac{1}{4}$ inch guide wire, the other the $\frac{3}{4}$ inch guide wire for a 24 inch target screen distance. By placing the scales on the side from the central line of the screen (Fig. 5) and by using the apparatus at a certain distance the screen is raised or lowered until the scale corresponds to this distance. The scale gives the distance from the end of the projecting piece (*b*) to the foreign body. See text.

cross-bar is so placed that the projecting piece (*b*) is on the patient's skin directly over the foreign body in the central ray of the focal spot.

The next step is to construct a scale. Select a convenient distance between the table top and screen, e.g., 12 inches, and then determine the focal spot screen distance. If this is unknown, a simple method to obtain this is as follows: Take a metallic marker—a needle stuck in a piece of adhesive tape is excellent for this purpose. Take the above apparatus and lower the imposed on the shadow of the $\frac{1}{4}$ inch distant wire on the screen. From Figure 1 it can be seen that similar triangles are formed and the mathematical relationship below holds. *FBM* (the distance of the foreign body to the skin marker *M*): *FSO* (the focal screen distance) = *MM'* (the distance between the $\frac{1}{4}$ inch wire markers): *OB* (the shift on the screen where the shadows are superimposed). *OB* can be measured directly on the screen and the equation solved for *FSO*, the focal screen distance.

To obtain the central line of the focal

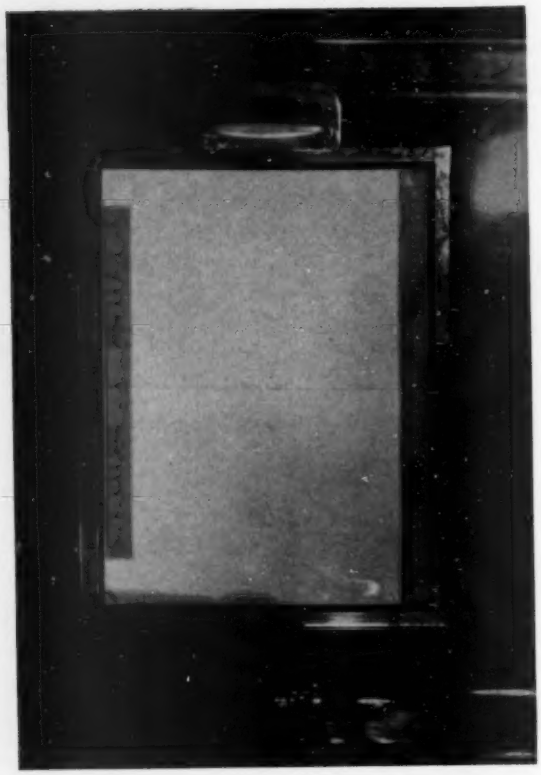

Fig. 5. The scales are placed on either side of the central ray. The scale is read directly during roentgenoscopy by the reflected light from the screen. The scales are for a 24 inch target screen distance, one calculated for $\frac{1}{4}$ inch guide wire, the other for the $\frac{1}{4}$ inch guide wire.

spot place a ruler with a metallic edge at right angles to the table top. Raise and lower the screen and shift the screen until a position is reached where the shadow of the ruler edge is stationary when the screen is raised or lowered. The center line of the screen is not necessarily in the center of the diaphragm openings.

After the focal spot screen distance *FSO* is determined, the shift *OB* for any depth can be calculated from the equation above. Practically this is done in $\frac{1}{4}$ inch steps, as the intermediate positions can be determined by interpolation.

The calculation is done for the first 4 inches using the $\frac{1}{4}$ inch wire guides. For the next 4 inches it is better to use the 1 inch wire guide as the distance of the shift *OB* would become small and hence difficult to measure practically. Hence two scales are used, one using the $\frac{1}{4}$ inch wire, the other the 1 inch wire.

Scales calculated for a 24 inch focal screen distance are illustrated. If desired, these scales can be cut out and transferred to the screen from the central line, as illustrated in Figures 3, 4 and 5. Place the apparatus over a marker, e.g., a needle at a certain distance, e.g., 2 inches from the end of the projecting piece (*b*) to the needle. Center the shadows of the end wire with the projecting piece (*b*) over the central line of the screen and center this over the needle. Then shift the tube and screen until the needle shadow *M* and the $\frac{1}{4}$ inch wire shadow *M'* superimpose. Raise or lower the screen until the superimposed shadows of the needle and $\frac{1}{4}$ inch wire *M'* correspond to 2 inches on the scale. This will give a 24 inch focal spot screen distance. It is useful to measure and cut a wooden block of this distance from the table top, and all that is necessary to obtain the same focal spot screen distance is to bring the screen down until it reaches the top of the wooden block.

The two scales can be transferred to some clear roentgen film using india ink and fixing them to the edge of the screen (Fig. 4 and 5). This will not interfere with subsequent roentgenoscopic procedure and will enable one to read directly the scale during the examination by the reflected rays from the screen. It is also more convenient to duplicate the scales on each side of the central line.

A scale can also be made directly by merely raising the cross-bar at definite distances from the needle and noting the distance of the shifts with a skin marking pencil.

To localize the depth of a foreign body is now a simple matter taking a few minutes and is read directly during the roentgenoscopic examination.

Step 1: Place the patient in the surgical position, e.g., the position at which he would be operated upon. Locate the foreign body and select a certain part of it and

center this with the center line of the screen.

Step 2: Place the apparatus upon the table and lower the cross-bar until the projecting piece (*b*) is upon the skin. Then center the shadow of the foreign body with the shadow of the end wire of the cross-bar with the center line of the screen until they are superimposed.

Step 3: Shift the tube and screen so that the same position of the foreign body chosen will be superimposed on either the shadow of the $\frac{1}{4}$ inch wire or the $\frac{3}{4}$ inch wire, and read the depth directly from the scale on the screen.

It can readily be seen that the shift of the tube and screen can be done either at right angles to the long axis of the tube or parallel to the table. It is only necessary to change the position of the wires in reference to the cross-board and the position of the scales.

The author wishes to express his appreciation to Dr. Foster J. Curtis, Chief Medical Officer, for his kind cooperation.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

Hemphill, James E. A simple technic for cerebral arteriography. *Radiology*, Oct., 1942, *39*, 432–436.

A simple technique for cerebral arteriography is described and illustrated with roentgenograms. The internal carotid on the side of the suspected lesion is exposed through a small incision under intravenous anesthesia. A silk thread is passed under the artery to stabilize it during the injection of the contrast medium. On the roentgen table the side opposite the exposed artery is placed toward the film which is in position in the Bucky carriage. Two other films are available near by. A needle attached to a syringe containing 20 cc. thorotrast is inserted into the exposed artery. Exposure is delayed for two or three seconds after blood enters the syringe and then the first film is exposed. This shows the outline of the internal carotid to the level of the anterior, middle and posterior cerebral vessels. The two subsequent films are exposed as quickly as possible. The three exposures can be completed in eleven to twelve seconds. The second film shows most of the smaller branches of the anterior, middle and posterior cerebral arteries. The third shows the smallest cerebral arteries, the venous pools and usually the larger arteries still filled with the contrast medium. The wound is closed while the films are being developed. The hemorrhage from the puncture wound in the artery can be controlled by sponge pressure.—*Audrey G. Morgan.*

Dandy, Walter E. Intracranial arterial aneurysms in the carotid canal; diagnosis and treatment. *Arch Surg.*, Sept., 1942, *45*, 335–350.

A case of large aneurysm of the carotid artery beginning in the carotid canal and extending into the skull is described. The patient was a woman of fifty-two whose symptoms began with diplopia followed by severe pain in the right eye, piercing headache in the right frontal region, nausea and vomiting, and ptosis. The most important signs of an aneurysm in this region are palsy or paralysis of the third nerve and severe pain in the affected eye or frontal region. These signs may also occur, however, in intracranial aneurysms of the internal carotid or even of the posterior communicating artery. But when there is also subjective or objective involvement, or both, of the first and second branches of the trigeminal nerve, as in this case, the diagnosis of aneurysm in the carotid canal is almost certain. Angiography has been used for the diagnosis of these tumors but the author advises against it, as it is not free of danger.

In this case operation was performed successfully by establishing adequate collateral circulation in the circle of Willis by preliminary partial ligation of the carotid in the neck and then trapping the aneurysm by placing a silver clip on the internal carotid inside the skull and ligating the artery with silk in the neck. The author had previously used this method in smaller aneurysms but feared it would not be effective in one as large as this. Pulsation of the aneurysm stopped within five minutes. Two months later the patient was free of all symptoms except that the exophthalmos had not decreased.

A table is given showing the outstanding features of 35 similar cases reported in the literature. Among the 7 patients operated on 1 died, a mortality of 14 per cent. The other 6 patients recovered, the longest interval since operation being three and a half years.—*Audrey G. Morgan.*

Dyke, Cornelius G. Acquired subtentorial pressure diverticulum of a cerebral lateral ventricle. *Radiology*, August, 1942, *39*, 167–174.

In the condition described in the title a part of the medial portion of the atrium of a lateral ventricle is pushed medially and caudally through the incisure in the tentorium and forms a sac filled with fluid beneath the tentorium. If care is not used in diagnosing this condition it may be mistaken for an enlarged fourth ventri-

cle displaced rostrally, which would lead to mistaken diagnosis and treatment.

Three cases of this form of diverticulum are described, in 2 of which the increased intracranial pressure that led to the condition was caused by brain tumors. The first was a case of astrocytoma of the mid brain with stenosis of the aqueduct in a man aged twenty-nine, the second a case of parasagittal meningioma in a woman aged forty-six and the third a case of an obstructing scar in the aqueduct of Sylvius in a girl aged sixteen. In 2 cases the diagnosis was made from pneumencephalograms, which are reproduced.

The nature of the wall of these diverticula is not known except that it probably consists of a thin layer of brain tissue and leptomeninges and may be lined with ependyma. A detailed discussion is given of the site of the brain defect in these cases, the way in which it is brought about and the importance of its recognition. —*Audrey G. Morgan.*

ANDERSON, FRANK M., and ADELSTEIN, LEO J. Ganglion cell tumor (ganglioglioma) in the third ventricle. *Arch. Surg.*, July, 1942, *45*, 129–139.

Since interest in the subject has increased in recent years it is known that ganglion cell tumors of the brain are not so rare as they were formerly thought to be.

The authors describe a case of ganglioma of the third ventricle in a boy nine years of age seen at the Los Angeles County Hospital. He had shown gradual impairment of vision for three years with progressive development of headache, somnolence and vomiting. Mind and personality were not affected. There were no disturbances of gait and no tendency to stagger. Ventriculography showed bilateral symmetrical internal hydrocephalus with no air at all in the third ventricle. Operation showed a large ganglioma of the third ventricle which was completely removed and the patient made an uneventful recovery

There is no pathognomonic clinical picture in these tumors and ventriculography should be performed in any case in which there are good reasons to suspect such a tumor. The technique of the surgical operation is described. A frontal approach through the dilated lateral ventricle is indicated unless the tumor is in the posterior part of the ventricle when it must be approached from above through the corpus callosum.—*Audrey G. Morgen.*

NECK AND CHEST

DICKES, R., KNUDSEN, A. F., and FRANCO, S. C. Multiple polyps of the esophagus. *Arch. Int. Med.*, July, 1942, *70*, 121–131.

A case of gastrointestinal hemorrhage from polyps of the esophagus is described. This is an extremely rare condition and only one other case is described in the literature.

The patient was a girl of ten when first admitted to the hospital suffering from hemorrhage. The diagnosis at that time was thought to be Banti's disease with hemorrhage from esophageal varices. Splenectomy controlled the hemorrhage temporarily but, in spite of the fact that the liver did not show evidence of permanent damage and portal pressure was not increased, the hemorrhages recurred. The taking of a biopsy specimen was followed by very severe hemorrhage. The specimen showed many blood spaces in the thickened squamous epithelium.

Finally five years after the beginning of the disease and after repeated esophagoscopic examinations a diagnosis of polyps of the esophagus was made. The polyps were treated by fulguration and the patient has been free of hemorrhage since that time.

There is some difficulty in the differentiation of esophageal polyps and esophageal varices after giving barium sulphate. Both appear as translucent areas in the lumen of the esophagus. Varices, however, show momentary variations probably resulting from peristalsis, while these polyps showed no such variations. This may prove a useful point in differential diagnosis. —*Audrey G. Morgan.*

TAYLOR, HENRY K., and SHULMAN, ISIDOR. Cardio-angiography. *Radiology*, Sept., 1942, *39*, 323–333.

Cardio-angiography is a method of examining the heart by injecting a contrast solution and making roentgenograms after periods calculated from the results of circulation tests. In 13 of 15 cases the circulation time of the right heart was approximately five seconds. That of the left heart is less constant.

This article is based on the study of a group of 15 patients, 4 of whom had rheumatic heart disease, 1 coronary thrombosis, 2 hypertension, 2 arteriosclerotic heart disease, 3 syphilitic heart disease and 3 bronchial asthma. A 70 per cent diodrast solution was used as contrast medium. Roentgenograms of normal hearts and

these various pathological conditions show a number of differences in the heart outline from that described in the classical texts.

This method should not be used in all heart cases, but only in those with unusual diagnostic problems. It is valuable in the diagnosis of pericardial effusions, arterial or ventricular aneurysms, constrictive occlusion and thrombosis of the superior vena cava and in differentiating cardiovascular from other mediastinal lesions. There were no serious reactions in any of the author's cases. The method is not recommended in patients who are acutely ill or who have severe decompensation or respiratory difficulty.—*Audrey G. Morgan.*

Peery, Thomas M. Incomplete rupture of the aorta; a heretofore unrecognized stage of dissecting aneurysm and a cause of cardiac pain and cardiac murmurs. *Arch. Int. Med.*, Nov., 1942, *70*, 689–713.

Incomplete rupture of the aorta is really a preliminary stage of dissecting aneurysm. Approximately the inner two-thirds of the intima are ruptured, the rupture usually occurring in the ascending part of the aortic arch at or near one of the commissures. The essential predisposing cause of incomplete rupture is severe hypertension continued for a long time. The precipitating cause may be strain or exertion.

Illustrative cases found on autopsy are described, with anatomical drawings and electrocardiograms. If dissection does not occur and the tear is well above the aortic commissures healing usually takes place. But if the rupture is at or just above a commissure and transverse to it, it usually causes aortic insufficiency and possibly death from congestive heart failure.

There are some points that may possibly be helpful in making an antemortem diagnosis of this condition. If a patient has had hypertension for a long time and suddenly has an attack of suffocation and dyspnea, which may or may not be accompanied by substernal pain, and there are systolic or diastolic murmurs or both, which had not been present before, it is suggestive of incomplete rupture, especially if the murmurs are harsh or rasping.

If the patient is seen for the first time during an acute attack and there are no murmurs a mistaken diagnosis of coronary thombosis may be made. The sudden onset of symptoms may suggest rupture of an aortic valve cusp.

In dissecting aneurysm the pain is usually more severe and the severity and location of the pain change as the dissection progresses. If the patient is first seen during heart failure caused by aortic insufficiency diagnosis is very difficult. If the symptoms begin suddenly, pressure is very high and there are no signs of syphilitic or rheumatic infection it may be possible to diagnose incomplete aortic rupture as the cause of the insufficiency.

Roentgen examination usually shows widening of the aortic arch but no signs pathognomonic of incomplete rupture.—*Audrey G. Morgan.*

Tager, Stephen N. Use of over-penetrated film technic in diagnosis of cavities. *Radiology*, Oct., 1942, *39*, 389–394.

The author emphasizes the value of the over-penetrated film technique in the demonstration of cavities in tuberculosis. He has used a very simple method of over-penetrated film technique that is available to any roentgenologist while laminagraphy is not available because of its prohibitive cost. In cases that show only a homogeneous veiling with the routine technique the over-penetrated technique may definitely reveal the presence of cavities. A series of routine and over-penetrated films is given illustrating this point.

The factors for the routine technique were 75–85 kv., 100 ma., time 1/10 second, distance 72 inches, rectification mechanical. For the over-penetrated film 50–60 kv., 100 ma., time ½ to 1 second, distance 40 inches, Bucky-grid ratio 5–1, rectification mechanical. It is to be noted that the Bucky grid is used. The term Bucky film is synonymous with over-penetrated film.—*Audrey G. Morgan.*

BLOOD AND LYMPH SYSTEM

Erf, L. A. Clinical studies with the aid of radiophosphorus. II. The retention of radiophosphorus by tissues of patients dead of leukemia. *Am. J. M. Sc.*, April, 1942, *203*, 529–535.

A study was made of the concentration of radiophosphorus in 32 patients dead of leukemia, 1 of neuroblastoma and 1 of chloroma. The technique of making the determinations is described and a table given showing the amount and dates of the doses of P^{32} given and amounts retained in the various tissues and organs in

microcuries per gram of wet weight of tissue assayed.

Because of the innumerable differences in age, weight, basal metabolic rate and diet of the patients, differences in the types of leukemia and in the amounts and routes of administration of the radiophosphorus given, it is hard to draw definite conclusions, but in general this work confirms that previously done on mice and shows that the greatest concentration of radiophosphorus is in the tissues which are most frequently infiltrated with leukemic cells.—*Audrey G. Morgan.*

PASCUCCI, LUCIEN M. Chronic leukemia; statistical study of symptoms, duration of life, and prognosis. *Radiology*, July, 1942, *39*, 75–80.

This study is based on 64 patients with myeloid and 64 with lymphatic leukemia treated by roentgen irradiation at the Presbyterian Hospital in New York between 1919 and 1939. Cases in which the duration of life was less than four months from the beginning of symptoms were considered acute and are not included in the study. All patients with chronic leukemia were given roentgen treatment. Irradiation of the spleen was used in all except two cases in which general irradiation of the body was given. At first medium voltage was used, later high voltage treatment: 200 kv., 8 or 25 ma., 50 cm. distance and 1 mm. copper and 1 mm. aluminum filter. The skin over the spleen was given 25 to 50 r once or twice a week for several weeks, after which a dose of 75 r was given once a week. If there were enlarged peripheral lymph nodes they were given 50 r, which after one or two weeks was increased to 100 r.

Weakness and loss of weight occurred about one and one-half times as frequently in patients with myeloid leukemia as in those with lymphatic leukemia. There was a high incidence of splenomegaly in the myeloid form and almost as high in the lymphatic. The incidence of fever and hemorrhage was about the same in the two types. Retinitis was four times as frequent in the myeloid as in the lymphatic type.

The duration of life in chronic leukemia varies from two to four years according to reports in the literature but the authors found an average of 2.5 years in myelogenous and 2.8 years in lymphatic leukemia. It has not been definitely proved that roentgen treatment lengthens life but it certainly brings about remissions during which the patient may lead a normal and efficient life. And occasionally it seems life is prolonged.

An early fatal outcome is likely if the platelet and red cell counts are very low when the patient comes for treatment. Graphs are given showing the relation of survival to red blood count, platelet count and primitive cell count in the two types of leukemia. The more chronic the disease, the greater the life expectancy. The question of whether acute and chronic leukemia are really the same disease is discussed.—*Audrey G. Morgan.*

VIETA, J. O., FRIEDELL, H. L., and CRAVER, L. F. Survey of Hodgkin's disease and lymphosarcoma in bone. *Radiology*, July, 1942, *39*, 1–15.

The authors present data and conclusions based on a study of 38 cases of Hodgkin's disease and 15 cases of lymphosarcoma, in all of which there were roentgen signs of changes in the bones. A postmortem study was made of 47 cases of Hodgkin's disease and 54 cases of lymphosarcoma. There were bone changes in 49 per cent of the former and 29 per cent of the latter. A roentgen review of 257 cases of Hodgkin's disease showed 14.8 per cent with bone involvement and a similar study of 213 cases of lymphosarcoma showed bone lesions in 7 per cent. The incidence of bone changes in both these series is probably lower than the actual facts due to inadequate examinations. The bones most frequently affected were those of the pelvis, the vertebrae, ribs and femora; in most cases more than one bone was involved.

Involvement of bone in both these diseases takes place either by direct extension from affected lymph nodes or through the blood circulation. The most frequent clinical signs of bone involvement were localized pain and tenderness but sometimes there were also tumors around the edges of the bones and neurological changes caused by extension of vertebral lesions into the spinal canal. In both diseases a high serum phosphatase content seems to indicate involvement of bone. This change was found most frequently in cases in which the bone changes were osteoplastic. In Hodgkin's disease 57 per cent of the cases showed a combination of osteoplastic and osteolytic changes, while in lymphosarcoma the changes were entirely osteolytic in 85 per cent of the cases. The character of the bone lesions is

different in the two diseases but not sufficiently so as to make a sharp differentiation between the two types possible.

The bone changes may occur at any time in the course of the disease. They are more apt to be terminal in lymphosarcoma as in this disease they occurred in 63 per cent of the cases in the last third of the course of the disease while in Hodgkin's disease they occurred in two-thirds of the cases earlier than that.

Irradiation with moderate doses often brings about marked improvement in the symptoms of Hodgkin's disease and in some cases restores normal bone structure. There was sometimes clinical improvement in lymphosarcoma also but death occurred so soon after the beginning of the bone changes that it was impossible to study the changes in the bones brought about by the treatment.—*Audrey G. Morgan.*

GENERAL

CAHILL, GEORGE F., MELICOW, MEYER M., and DARBY, HUGH H. Adrenal cortical tumors; types of nonhormonal and hormonal tumors. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 281–305.

This is a report of the adrenal cortical tumors which have been seen at the Squier Clinic (Department of Urology and Biochemistry, College of Physicians and Surgeons, Columbia University).

Tumors of the adrenal may be classified as follows:

(1) Tumors from the connective tissue, blood vessels, lymphatics, or nerve structures such as may occur in any gland and are not peculiar to the adrenal. These occur rarely.

(2) Tumors derived from the medulla which are peculiar to that tissue and are derived from the ectodermal sympathetic nerve system. They may be of the basic nerve cell, or sympathoblastoma; from the sympathetic ganglion or ganglioneuroma; from the pheochromocytes or paragangliomas. The first two are seen more frequently in children and are more common than the paragangliomas which occur more often in adults.

(3) Tumors originating from the adrenal cortex are mesodermal glandular tumors and are, cytologically, hyperplasias, adenomas, or carcinomas.

Accessory adrenals occur and are called Marchand's bodies. These are made up of cortical tissue and they are rare.

The Adrenal Cortex and Tumors of the Adrenal Cortex. The adrenal cortex has been definitely established as the elaborator of hormonal substances some of which are necessary for life maintenance. Chemically, these hormonal substances have been shown to be sterol lipoids. The adrenal cortical tumor occurs relatively infrequently. The clinical manifestations of these tumors may be as follows:

(1) No recognizable hormonal changes
(2) Changes due to excess androgens
  a. In female child toward adult masculinity
  b. In female adult toward masculinity
  c. In male child toward adult masculinity
(3) Changes due to excess estrogens
  a. In adult male toward femininity
(4) Changes due to excess androgens and other steroids
  a. Cushing's syndrome with associated sexual changes (mostly in females)
(5) Changes due to excess other steroids related to metabolism
  a. Cushing's syndrome without sexual change (in male and female)

Diagnosis and Treatment of Adrenal Cortical Tumors.

(1) The status of the adrenals may be adequately shown by air insufflation roentgenograms.

(2) Removal of adrenal tumors by the transperitoneal route is surgically best.

(3) Acute adrenal deficiency occurs only in those patients with symptoms described as Cushing's syndrome.

(4) The therapy of acute adrenal deficiency is similar to that which is used in the acute deficiency crisis in Addison's disease.

(5) Histologically, the tumors that produce the hormonal syndromes have cytoplastic lipoid vacuoles in amounts comparable to the symptoms.—*Mary Frances Vastine.*

ROENTGEN AND RADIUM THERAPY

BUTLER, FRANK E., and WOOLLEY, IVAN M. The present status of roentgen therapy in chronic paranasal sinusitis. *Radiology*, July, 1942, *39*, 69–74.

The authors first reported a series of 100 cases of chronic sinusitis begun in 1929; their results were greeted with a great deal of skepticism by

rhinologists and the method severely criticized. Now, however, they report 450 cases treated up to 1934 with 36 per cent of the patients free of symptoms, and 55 per cent improved. Compilation of the statistics of six observers shows an average of 33 per cent symptom-free, 41 per cent improved and 26 per cent unimproved. They have never yet seen any harmful results from the treatment and it does not cause fibrosis which would interfere with later operation as was predicted.

Illustrations are given of the masks used to protect the part of the face that is not to be irradiated. The authors prefer the single massive dose technique though equally good results have been obtained by other workers with the fractional dose method. They believe more depends on the skill of the operator than on the technique. They find a 200 kv. technique superior to a 120 kv. one because the greater filtration gives a wider margin of freedom from skin reactions; it also makes it possible to give a greater amount of radiation through the sphenoid sinuses. They now use 200 kv., 20 ma., through the equivalent of 0.75 mm. copper and 3 mm. aluminum filter at a distance of 50 cm., half-value layer 0.02 mm. copper; a dose of 300 r measured in air is given on each side and the patient told to return in six to eight weeks if he has further symptoms.

Roentgen treatment is not successful in allergic sinusitis. The best results are seen in chronic infections with a markedly thickened membrane and a small central pneumatized cavity. —*Audrey G. Morgan.*

FRIEDMAN, MILTON, and FINKLER, R. S. The treatment of sterility with "small dose" x-ray therapy. *Am. J. Obst. & Gynec.*, May, 1942, *43*, 852–857.

A case report is given which strongly suggests that small dose roentgen therapy can favorably affect sterility due to ovarian deficiency. In 1935, the patient's sterility was accompanied by irregular menses, possible estrogen deficiency (as evidenced by bio-assays), and faulty luteinization (as evidenced by endometrial biopsies). The first course of small dose roentgen therapy was followed by normal menses and two pregnancies within nine months. The first pregnancy was accidentally interrupted by a pre-menstrual biopsy which removed a twelve to fourteen day old embryo. The monthly cyclical bleeding was not thereby disturbed. The second pregnancy resulted in the delivery of a normal child at full term. Four years later, the clinical picture of ovarian deficiency returned in a more severe form, as evidenced by amenorrhea and absent luteinization. A second course of small dose roentgen therapy was followed by restoration of normal menses and a third pregnancy with delivery of a normal child.

Both courses of roentgen therapy were the same. Four treatments were given over a seven to ten day interval. A total dose of 80 roentgens (measured with back scattering) was given to the pituitary gland and 80 roentgens was given to each ovary.—*Mary Frances Vastine.*

SEWELL, ROBERT L., DOWDY, ANDREW H., and VINCENT, JAMES G. Chemotherapy and roentgen radiation in Clostridium welchii infections. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 361–367.

Fifteen patients with "gas gangrene" have been seen in the Strong Memorial Hospital, Rochester, N. Y., since 1937. The authors' work is based on the experience with these 15 cases together with the study of 255 dogs in which they produced *Clostridium welchii* infection experimentally.

Clinical clostridium infections range in severity from relatively benign infections to more fulminating and malignant types. In this work the criteria for calling cases "gas gangrene" were: (1) positive cultures of *Clostridium welchii;* (2) spreading infection; (3) gas in the tissues, and (4) discoloration and edema of the involved structures.

The summary of the findings is outlined as follows:

(1) Many of the virulent infections caused by *Clostridium welchii* are more of the nature of a cellulitis than a gas gangrene.

(2) Radical surgery is recommended only in cases of severe vascular damage.

(3) Sulfanilamide or roentgen irradiation, when used alone, exerts a beneficial effect on the course of the disease, and to about the same degree. Sulfathiazole is probably somewhat more efficacious. Sulfadiazine is superior to any other drug or method of treatment (experimental proof only).

(4) The combined use of both sulfadiazine and roentgen irradiation has given poorer results experimentally than has the use of either separately.

(5) *Clostridium welchii* antiserum is of some

efficacy only when additional methods are used to combat the local lesion.

(6) Conservative surgery coupled either with a sulfonamide or with roentgen irradiation is the treatment of choice. At present, conservative surgery plus sulfadiazine is recommended. —*Mary Frances Vastine.*

Cosbie, W. G. The complications of irradiation treatment of carcinoma of the cervix. *Am. J. Obst. & Gynec.*, Dec., 1941, *42*, 1003–1008.

The hope of increasing the salvage rate of patients suffering from carcinoma of the cervix depends on carrying radiation treatment to a point sometimes beyond the tolerance of the adjacent tissues. This is true because 65 to 70 per cent of patients suffering from carcinoma of the cervix do not come for treatment until the disease has progressed to Stage III or IV. Two types of reaction are met with: (1) the acute or inflammatory, and (2) the late or fibrotic which may pass on to a state of necrosis.

This study includes 320 patients treated for carcinoma of the cervix during the years 1936 to 1940 inclusive. The complications which can occur may be outlined as follows:

(1) Irradiation sickness (with cystitis and proctitis) is a fairly frequent development during or immediately following treatment.

(2) Cystitis is much less frequent when a self-retaining catheter keeps the bladder empty during radium treatment. Distortion of the base of the bladder with displacement of the ureteral openings is commonly observed.

(3) Carcinomatous infiltration of the bladder was found a number of times on cystoscopic examination when its presence had not been suspected. Seventeen of the 14 autopsies held on patients in this series showed ureteral obstruction, hydroureter and hydronephrosis or pyonephrosis. In every case, the obstruction was due to malignant infiltration of the cellular tissue.

(4) Fever and infection during the course of treatment may cause much concern. Out of 47 patients in the study with positive hemolytic streptococcus cultures, 25 developed some complication subsequently such as excessive pyrexia, vaginitis, pyometria, cellulitis, thrombophlebitis, pelvic abscess, peritonitis or septicemia.

(5) Proctitis is encountered frequently but it is a condition which ordinarily disappears within a few weeks. The typical late irradiation lesion is rectal ulceration. Two types have been described: (*a*) primary chronic ulceration of the rectum, and (*b*) an extrinsic type which is characterized by massive perirectal cellulitis and stenosis of the rectum as a result of which ulceration develops. The warning is given that symptoms simulating intestinal obstruction several months or years after irradiation may well be due to stricture rather than metastases.

(6) Spontaneous fracture of the neck of the femur following roentgen treatment of carcinoma of the cervix can occur but none of the patients in the series under discussion experienced this complication.—*Mary Frances Vastine.*

Desjardins, Arthur U. Problems in roentgen therapy for Hodgkin's disease and lymphosarcoma. *Radiology*, July, 1942, *39*, 16–26.

Often the maximum results are not obtained in treating Hodgkin's disease or lymphosarcoma with roentgen rays. This may be because the rays have not been applied to the fields where the cause of the trouble is really located; it may be due to improper time distribution of the treatment, wrong quantity or quality of rays, pathological factors or clinical factors that depend on the stage of the disease. There is a general tendency to direct treatment to the cervical, axillary and inguinal lymph nodes but there may be involvement of the mediastinal or abdominal or retroperitoneal nodes and the fields must be selected accordingly. Irradiation of the abdomen affects the leukocytes more than that of any other part of the body so the leukocyte count must be watched very closely during abdominal irradiation.

From the author's experience with more than 2,000 cases of lymphoblastoma he believes that irradiation at 140 kv. is more effective than that at 200 kv., provided the treatment is applied to the correct anatomical fields and excessive doses are not given. This is true even of mediastinal and retroperitoneal lymphoblastoma.

The doses required are not as large as those for epithelioma. Therefore protracted fractional irradiation is not necessary. The best long-terms results are obtained by giving each field in one or two days as large a dose as can be tolerated without injuring the skin, usually 550 to 600 roentgens, surface dose measured in air. The fields are treated successively one at a time until all have been given the required dose. This depends on the extent of the involvement and the tolerance of the patient.

Failure may be due to giving too small doses at each treatment or leaving too long intervals between treatments. Or it may be due to pathologic factors such as beginning treatment in the terminal stage of the disease. Special problems may be created by lymphoblastomatous infiltration of the skin, subcutaneous tissues, abdominal organs, rectum, orbit, meninges, ureter or kidney or suprarenal glands or erosion of a bronchus, which may result in fatal hemorrhage if not treated in time. The symptoms of these various types of infiltration are discussed and the importance of early detection and treatment stressed.—*Audrey G. Morgan.*

RANDALL, LAWRENCE M., LOVELADY, SIM B. and SLUDER, FLETCHER S. Radium in the treatment of uterine bleeding caused by benign lesions. *Am. J. Obst. & Gynec.*, March, 1942, *43*, 377–387.

Complete follow-up records were studied on 196 women who experienced atypical uterine bleeding (menorrhagia and/or metrorrhagia) caused by benign conditions, five or more years after treatment by radium or roentgen rays. One hundred seventy-five patients were treated with radium and 21 with roentgen rays. The bleeding was satisfactorily controlled by the initial treatment in 134 (68.4 per cent). In 13 cases subsequent irradiation was necessary before bleeding was controlled. Thus good results were obtained in 147, or 75 per cent. Of the 125 patients who were forty years of age or younger and treated with radium, 21 (16.8 per cent) established a fairly normal menstrual cycle after radium therapy. The percentage of good results from all types of radium treatment increased with the age of the patients. As the age of the patient increased, the dose of radium administered tended to increase. The authors believe that for patients more than forty years of age without fibroids or when fibroids are present but the uterus is not enlarged to more than its size after three months of pregnancy, preliminary dilatation and curettage for diagnosis followed by intrauterine application of a menopausal dose of radium or the administration of roentgen therapy are excellent forms of treatment for atypical uterine bleeding. For the group of patients who are from thirty to forty years of age, the choice of treatment is made with more difficulty. To one who is surgically minded, failure of about 25 per cent from irradiation in the entire series would indicate that hysterectomy accompanied by a 98 to 99 per cent possibility of cure is preferable.

The dosages of radium and roentgen radiation given these 196 patients are indicated in tables included in the article. Of the patients forty years of age or less without myomas (79), some received less than 500 mg-hr., others 500 to 1,000 mg-hr. and another group 1,000 to 2,000 mg-hr. or more. The doses of radium in the 35 patients forty years of age or less with myomas varied from 500 mg-hr. to 1,000–1,200 mg-hr. or more. The patients who were more than forty years of age with or without myomas (47) received 1,000–1,200 or more mg-hr. of radium. The total roentgen-ray dosage administered to 21 patients varied from 700 to 1,760 r. —*Mary Frances Vastine.*

STEVENS, ROLLIN H. Radium poisoning. *Radiology*, July, 1942, *39*, 39–47.

A case of radium poisoning is reported in a man of thirty-six, a commercial photographer who had always been well except for night sweats, which he had had since youth. In the spring of 1924 he first noticed enlarged lymph nodes on both sides of the neck and in the axillae and groins. Histological study of a piece excised from one of these nodes showed Hodgkin's disease.

Roentgen treatment was begun on July 29, 1924, and the size of the nodes decreased. But on January 27, 1925, roentgen examination of the chest showed continued enlargement of the mediastinum and it was concluded that the injection of radium was justifiable in order to cure the Hodgkin's disease. On January 21, 1925, 20 micrograms of radium chloride was injected. Injections were continued up to November 24, 1930. During this period of five years and nine months 440 micrograms was given. On June 4, 1938, a dentist discovered an area of necrosis in the jaw bone. This necrosis soon involved the right side of the mandible and the jaw became fixed. Suffering was severe for months. Measurements showed that the bone was radioactive. The patient was put on a calcium-free diet, thyroid extract and ammonium chloride which stopped the pain and resulted in great improvement. Subsequently he again suffered great pain; he was put on this treatment again and recovered again. Now after eighteen years of treatment he is well, is continuing his work and does not show any signs of Hodgkin's disease. His blood picture has re-

mained normal throughout the years except that occasionally there has been 4 to 5 per cent eosinophils. There is no sign of malignancy. He is happy and contented and says he would have taken the treatment for the cure of the Hodgkin's disease even if he had known it would caused the radium poisoning. The interest of this case lies in the fact that a known quantity of radium was given over a period of eighteen years. The amount retained was 11.4 micrograms out of 440 micrograms, or 2.5 per cent, more than enough for destructive action. —*Audrey G. Morgan.*

MISCELLANEOUS

Voegtlin, Carl. Trends in cancer research. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 561–564.

At the end of the nineteenth century, Claude Barnard's plea for controlled experimentation was applied to the cancer problem. As a result a large number of new facts have been discovered:

(1) The first important conclusion reached from experimental work was the high specificity of malignant cells. Each one of the many types breed true in vivo and in vitro.

(2) In 1915, the production of skin cancer in rabbits by the long continued topical application of coal tar was announced. This important discovery shifted the trend of cancer research into the field of chemistry.

(3) Recently some progress has been made in the study of the dietary factor in the production of induced tumors.

(4) The discovery of the artificial chemical carcinogens was a milestone in cancer research as it has made possible the experimental production of tumors under controlled conditions. It is firmly established that very large doses of female sex hormones acting over a long time can induce malignant tumors of the breast and uterus in mice and rats.

(5) Recently, claims of the carcinogenic action of cell free extracts of tissues and of urine from cancer patients have been made. Final proof of these claims must wait on more thorough investigation.

(6) Experiments with normal and regenerating rat livers and rat hepatomas make it appear that some of the important enzymatic activities of the tumor show marked decreases. This attribute may perhaps account for the specific tumor biology.

(7) A very great reduction in catalase activity in the hepatic tumor has been found while a lesser decrease has been noted in that part of the liver which is apparently normal in the tumor-bearing animal.—*Mary Frances Vastine.*

Rhoads, C. P. Precancerous lesions. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 549–551.

The common factor to be discerned in those changes which are known to tend to become neoplastic is probably an atrophy. This atrophy is probably due to some mechanism which prevents the normal life and reproduction of the affected cells.

A table in which are presented recognized examples of cancer which is known to follow the appearance of an atrophic process is reproduced:

PRECANCEROUS CHANGES

1. Skin
   - A. Keratoses of the atrophic skin
   - B. Xeroderma pigmentosa (atrophy)
   - C. Radiation dermatitis (atrophy)
   - D. Burn scars and lupus (atrophy)
   - E. Arsenic dermatitis (atrophy)
2. Kraurosis vulvae (atrophy)
3. Plummer-Vinson disease (atrophy)
4. Thyroid cancer after hypothyroidism
5. Cancer in undescended atrophic testicle
6. Cancer of liver on cirrhosis
7. Leucemia on aplastic anemia

Kinosita proved that the feeding of dimethylaminoazobenzene (butter yellow) to rats taking a diet of rice would regularly produce hepatic cirrhosis followed by hepatic cancer. This experiment was verified and further analyzed in the author's laboratory. It is observed that cancer recurs and grows in the face of a milieu which is incompatible with the life and growth of normal cells. Thus it has been proved that a concentration of a toxic breakdown product of butter yellow which will kill normal rat liver cells has no effect upon the growth and multiplication of liver cancer cells.—*Mary Frances Vastine.*

Lieberman, Louis M., Hodes, Philip J., and Leopold, Simon S. Roentgen therapy of experimental lobar pneumonia in dogs. *Am. J. M. Sc.*, Jan., 1941, *201*, 92–100.

The authors have pursued the study of roentgen therapy on experimentally produced lobar pneumonia for some three years, beginning just prior to the advent of chemotherapy. They continued these studies because of the very encouraging evidence.

Forty-five dogs were infected; 26 of these were treated with roentgen radiation; 19 serving as controls. The development of pulmonary disease was confirmed by roentgenoscopy and roentgenography.

The quality of radiation applied varied, including that produced by 80 kv. (5 mm. Al); that produced with 135 kv. (0.25 mm. Cu plus 1 mm. Al), and that produced with 200 kv. (0.5 mm. Cu plus 1 mm. Al).

Twenty of the animals were handled as group 1; half of these were treated with roentgen radiation, the other half serving as controls. The dosages varied from 70 to 120 r, one, two or three treatments being given at daily intervals. All of this group died. Group 2 included only 4 animals, 1 serving as control, the other 3 being treated with 135 kv. therapy. One of these survived (receiving 150 r the first day; 124 r the second). There were 21 animals in the third group, 8 of these serving as controls. The others were treated with 200 kv. therapy receiving treatments on the first, second or third days, varying from 107 to 240 r. Five of the 13 animals survived; those receiving, for the most part, the greatest dosage and treatments over the three days.

Pathologically, there were found pneumonic infiltrations, including congestion, edema, hemorrhage and neutrophile infiltration. Atelectasis was found in about one-half of the cases. The irradiated animals showed about the same degree of congestion and hemorrhage as found among the controls. However, those receiving roentgen radiation showed less edema, less atelectasis, a decrease in neutrophiles and a relative increase in round cell infiltration.

The authors speak of Korb's work which indicated that even with dosages as high as 22,000 r, using high voltage, there was no effect upon cultures of tubercle or colon bacilli; whereas, with rays generated at 50 kv. these bacteria were killed with smaller dosages. Korb also found that bacterial radiosensitivity increased markedly with increases in temperature. They also refer to those studies which indicate that irradiation increases the alkalinity of tissues and that an alkaline medium is more ideal for neutrophiles. Mention is also made of changes in the molecular structure of toxins as well as of allied proteins and the elevation of the globulin fraction (and the concurrent increase in antibodies). Reference is also made to Desjardins' theory of leukocytic degeneration by roentgen irradiation and the resultant stimulation to phagocytosis locally.—*A. A. de Lorimier.*

BEARD, J. W., TAYLOR, A. R., SHARP, D. G. and BEARD, DOROTHY. The nature of a virus associated with carcinoma in rabbits. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 509–513.

The isolation and properties of a specific material from virus-induced cottontail rabbit papillomas are described in this article. This material behaves biologically as does the virus responsible for the disease. It is a nucleoprotein obtainable in solutions of high homogeneity. Its physical properties with respect to sedimentation, electrophoresis, diffusion, and viscosity are those of a molecular substance. With respect to the physical state, this animal virus falls into the same category as that occupied by the macromolecular plant virus nucleoproteins.—*Mary Frances Vastine.*

ELLINGER, FRIEDRICH, and DAVISON, CHARLES. Changes in the central nervous system of goldfish irradiated in the depths of a water phantom. *Radiology*, July, 1942, *39*, 92–95.

Goldfish have been found suitable animals for the evaluation of the biological action of roentgen rays. In this article experiments are described in which goldfish were irradiated in the depth of a water phantom measuring 32×32×32 cm., which permits of maximum backscatter even at a depth of 10 cm.

Histologic examination of the brains of these goldfish showed marked changes, particularly in the medulla oblongata. The technique of the experiments and the histological findings are described. A comparison of the findings in one of the fish irradiated in the phantom and another irradiated at the surface showed that though the animals survived for about the same length of time there were more marked changes in the myelin sheaths of the fish irradiated in the depths of the phantom. There were greater changes also in the nerve cells of the medulla oblongata; there was almost complete destruction of the cell with a dose of 1,000 r in the depths while a dose of 1,250 r on the surface caused only slight decrease in the number of cells. In one fish an estimated body dose of about 340 r caused pigmentation and brain changes at a depth of 10 cm., though the minimal dose required to cause pigmentation on the surface is about 800 r in air.—*Audrey G. Morgan.*